LABYRINTH PAPERS

MACKENZIE

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BY

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THIS COLLECTION OF PAPERS IS DEDICATED TO MY FRIEND AND TEACHER,

PROFESSOR GUSTAV ALEXANDER.

VORWORT.

Es bereitet mir eine grosse Genugthuung, dem vorliegenden Werke meines Freundes und langjährigen Schülers Dr. George W. Mackenzie einige Worte vorausschicken zu dürfen.

Durch die Forschungsergebnisse der letzten Jahre hat die Klinik der Labyrintherkrankungen eine vollkommene Umgestaltung und ungeahnte Bereicherung erfahren. Die modernen Untersuchungsmethoden des Ohrlabyrinthes setzen uns instand Erkrankungsformen und Erkrankungsgrade des Labyrinthes zu diagnoszieren, die in früherer Zeit wegen der Geringfügigkeit der Initialsymptome oft lange unentdeckt geblieben sind.

Auch die Behandlung der Labyrintherkrankungen hat eine neue Gestaltung erfahren; es gilt diese sowohl für die leichteren Formen und die conservative Behandlung als auch für die operative Behandlung der schweren und komplizierten Formen der Labyrintheiterung.

Dr. George W. Mackenzie hat unter meiner Leitung mehrere Jahre sein ganzes Können und Studium der Frage der Labyrinthkrankheiten gewidmet. Ich habe im Laufe dieser Zeit nicht bloss erkannt, dass er das wissenschaftliche Material vollständig beherrscht, sondern dass er auch in vorzüglicher Weise imstande ist, Andere in die klinische Untersuchungs- and Behandlungsmethoden einzuführen und zu unterrichten.

Dr. George W. Mackenzie scheint daher in besonderer Form berufen und vorzüglich befähigt, ein Buch über die Klinik der Labyrinthkrankheiten zu verfassen.

Das Ziel, das er sich bei der Abfassung des Buches gesteckt hat, hat er vollständig und in vorzüglicher Weise erreicht.

Das Buch wird ebenso sehr dem Otologen, als auch dem Chirurgen, Nervenarzt, Internisten und Paediater ein willkommener Behelf sein.

Dr. Gustav Alexander.

a. o. Professor an der K. K. Wiener Universität
Vorst and der Ohrenabteile d. Algemein
Poliklinikin Wein.

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INTRODUCTORY REMARKS.

At the request of many friends in the profession, who have looked with favor upon my writings on the Labyrinth, I consent to the publication of the collection in this volume.

In June, 1908, when presenting my first paper on the subject of Labyrinth Suppuration, there was no intention of writing so extensively upon the Labyrinth; however, requests for others came in such rapid succession that it was not long before I realized that I was fairly launched in compiling a series of papers. The papers herein contained were witten for various Societies and Journals between June, 1908, and February, 1911.

Each of these papers practically comprises a chapter. The effort was to make each paper complete in itself and still make the various papers dove-tail into each other, and in the end to make the series complete by covering every phase of the subject of the Labyrinth and its diseases.

In a few instances, repetitions appear; however, where they have occurred the writer had thought them of sufficient importance to warrant the repetition.

I wish to take this opportunity of thanking my friend and teacher, Professor Adam Politzer, with whom I studied and worked as aspirant assistant in the general hospital of Vienna; and no less to Professor Gustav Alexander, with whom I studied and worked as aspirant assistant in the Polyclinic of Vienna. With the latter I spent two most profitable years, receiving valuable suggestions and encouragement.

THE DIAGNOSIS AND TREATMENT OF LABYRINTH SUPPURATION.

OUR knowledge of labyrinth suppuration, developed during the last few years, has revolutionized the science of otology. At the present time no one can pretend to practice otology who is not thoroughly familiar with this subject.

An understanding of labyrinth suppuration presupposes:

1. A knowledge of the anatomy of the inner ear and the relative positions of the semicircular canals in the skull.

2. A knowledge of the physiology, especially of the nonacoustic labyrinth, as determined by Ewald, Breuer, Crum-Brown, Mach, Kreidl, Alexander, Kubo, Barany, Stein, Krotoscheiner and others.

Upon the more recently gained knowledge of the physiology of the nonacoustic labyrinth, exact examination methods have been developed which make possible an exact diagnosis in practically every case of labyrinth suppuration.

The time allowed will not permit me to discuss in detail the physiology of the labyrinth; however, for practical purposes, enough of the physiology will be brought out while discussing the diagnosis and the methods of examination used. The symptoms and signs of acute labyrinth suppuration are briefly as follows:

1. Deafness of sudden onset.
2. Vertigo with nausea and vomiting.
3. Spontaneous rotatory nystagmus toward the sound side.
4. Disturbances of equilibrium.
5. Negative caloric reaction.
6. Diminished or negative reaction to turning.
7. Diminished or negative reaction to Galvanism.

We shall now discuss separately the above symptoms and signs in the order given.

1. Deafness of sudden onset.

Deafness is a constant symptom of acute labyrinth suppuration which differentiates it from other forms of labyrinthitis—diffuse serous labyrinthitis and circumscribed labyrinthitis. In the suppurative form of labyrinthitis the deafness is complete and permanent, while

in the other forms there are remains of hearing and the ultimate prognosis to hearing is good.

Since no one would care to remove a labyrinth where there are remains of hearing, nor on the other hand neglect to operate a suppurating labyrinth where the mortality without operation is above 70 per cent., the determination of deafness becomes a question of vital importance to the patient's hearing in the one instance and to the patient's life in the other.

Prior to the introduction of the new, small (a_1) fork of Bezold, the only errors made in the diagnosis of labyrinth suppuration arose from the uncertainty in the determination of deafness in a case where all other signs and symptoms were positive. At that time the best method for recognizing onesided deafness was the 2 and 3 meter speaking-tube.

I have examined cases of unilateral deafness with the speaking tube when it seemed impossible for the patient to hear from the good ear, which was completely closed with a moistened finger, and yet they were able to repeat whispered words. Experimentally I have examined cases where the labyrinth had been removed, and the patient was apparently able to hear whispered words from that side through the tube. The only explanation is that the sound must have been transmitted to the well ear. To avoid all possibility of transmission of sounds to the well ear, Bezold designed the new small (a_1) fork, which has the advantage of being heard very distinctly when held close to the ear and of not being heard at all at a distance of 7 or 8 inches (the width of the skull or the distance necessary to be traversed in order to be heard by the other ear). I have examined a number of cases of one-sided deafness, using both methods (the tube and the small a_1 fork) and the fork has invariably given more satisfactory results than the 3 meter tube. Beside these methods, the other functional hearing tests (Rinnè, Schwabach, Weber and the continuous chain of forks) will be an aid in diagnosing one-sided deafness.

2. Vertigo with nausea and vomiting.

In acute labyrinth suppuration the vertigo is very intense and invariably associated with nausea and vomiting. The vertigo is most pronounced at the onset of the process but gradually diminishes after two or three days; however, brief attacks of vertigo may be produced by rapid movements of the head for several weeks afterward. This

vertigo is characteristic and known as vestibular vertigo; it must be differentiated from ocular and central vertigo. Vestibular vertigo is characterized by certain subjective phenomena. The patient feels that the room and surrounding objects are moving about him in a circle around his axis of vision. The direction of this movement in a case of left labyrinth suppuration is the same as that made by the hands of a clock, and when the eyes are closed the patient has the sensation of falling to the right. In case of a right labyrinth suppuration the sensations are reversed.

Vestibular vertigo combined with sudden deafness speaks for some destructive process of the labyrinth, and when found in the course of chronic suppuration of the middle ear speaks for acute labyrinth suppuration.

For a further description of vestibular vertigo and its differentiation from the other types, see Panse's Work on *Schwindel,* 1902.

3. **Spontaneous rotatory nystagmus toward the sound side.**

This symptom is constantly present, and like the vertigo it is most pronounced upon the first day and gradually diminishes during the following three or four weeks, when it practically ceases. In exceptional cases it may be observed months afterward.

The vestibular nystagmus is due to the overbalance of impulses from the normal side (*Normal Tonus*). This normal tonus can be better understood when we produce artificially a condition similar to that found in labyrinth destruction. This experiment can be made upon normal people by applying the anode to one side (say the right side); this produces an anelectrotonus of the right side, suppressing the normal tonus. At the same time the normal tonus from the opposite or left side produces an overbalance of vestibular impulses from the left side and thereby a nystagmus to the left. Spontaneous nystagmus associated with labyrinth suppuration is most pronounced when the patient looks toward his well side (the side of his nystagmus); however, during the first few days it may be observed in all positions of the eyeball.

4. **Disturbances of equilibrium.**

These may be divided into two separate forms:—the early and the late. The *early* is more marked and is so intense as to amount to a loss of equilibrium, which compels the patient to seek a recumbent position.

The tendency of the patient is to fall towards the diseased side. This tendency is increased when the eyes are closed. Falling to the diseased side may be termed "Reaction falling"—an associated phenomenon of nystagmus.

The explanation of this phenomenon is as follows: In a case of right labyrinth suppuration the patient has rotatory nystagmus to the left, accompanied by the subjective sensation of falling to the left. In his attempt to correct this subjective sensation of falling to the left, he overcorrects and actually falls to the right (toward the diseased side).

The *late form* of disturbance of equilibrium is not accepted by some authors, including Barany; however I hold that there is a late form, as do also Alexander, Kreidl, Stein, Krotoscheiner, Frey and Hammerschlag. Recently I conducted a series of experiments upon a number of cases, of late one-sided labyrinth destruction and deaf-mutes (see "Klinische Untersuchungen über die labyrinthaeren Gleichgewichtstoerungen u. s. w." Arch. f. Ohren, Bd. 78, 1909.

I found that, without exception, every case of late one-sided labyrinth destruction showed positive signs of disturbance in equilibrium by the following tests: Rhomberg, the gait (forward, backward and to the sides, with open and closed eyes), standing on one foot, hopping forward and backward upon one foot, elevation on the Alexander Stein goniometer with the face forward, backward and to the two sides, with open and closed eyes.

5. **Negative caloric reaction.**

This examination method belongs to Barany (see his book on *Physiologie und Pathologie des Bogengangs Apparat*). 1908. Before the time of Barany, Breuer and others had experimentally produced vertigo in man and nystagmus of the head in animals, by the use of cold water syringed into the external canal; however, it remained for Barany to first adopt these reactions for clinical purposes.

The normal ear syringed with water colder than the body temperature, produces a rotatory nystagmus to the opposite side, lasting for a few minutes; whereas water warmer than the body temperature produces the opposite effect—rotatory nystagmus to the same side. These caloric reactions are explained by Barany as due to currents set up in the endolymph in the same manner as all other cur-

rents are produced in vessels containing fluid, when the temperature of any part of the fluid is changed.

One thing characteristic of the caloric reaction, worth mentioning here, is that the nystagmus may be changed in character and direction by changes in position of the head. As an illustration: If we syringe the right ear with cold water with the head in an upright position a rotatory nystagmus to the left is produced; now if we turn the head 90° to the left so that the head rests upon the left shoulder, the rotatory nystagmus to the left changes to a horizontal nystagmus to the right.

The caloric test is the most certain qualitative test of the nonacoustic labyrinth, and when negative speaks for destruction of function in the semicircular canals (a most valuable diagnostic sign of labyrinth suppuration). This method of examination is inapplicable in cases of obstruction of the external canal (atresia, stricture, severe otitis externa, furunculosus and large polyps), also in very acute inflammation with small perforations. In such cases it is well to be able to rely upon other methods of examination yet to be described.

6. **Diminished or negative reaction to turning.**

This is an important sign found in labyrinth suppuration. The turning test is made with the patient sitting on a revolving stool, fitted with a handle which the examiner uses to turn the patient.

Normally, turning to the right with the head in the upright position produces a horizontal nystagmus to the right during turning and a horizontal nystagmus to the left upon stopping. If the head is inclined forward 90° a rotatory nystagmus to the right is produced during turning and to the left after turning. This latter nystagmus is called the "After turning" or "After nystagmus."

If the head is inclined to the right 90° (that is the right side of the face upon the right shoulder) a vertical nystagmus upwards is produced during turning to the right and vertically downwards after turning.

If the position of the head is changed 180° to any of the above positions, the opposite nystagmi are produced. Inclination of the head 45° in either of the lateral positions produces oblique nystagmi. In short, it is possible to produce nystagmus in any desired direction by change of position of the head. It is even possible to produce mixed forms;

as an illustration: Upon bending the head backward 45° we get a horizontal nystagmus to one side combined with a rotatory nystagmus to the opposite side. For practical purposes, however we may limit our examinations to the horizontal and rotatory nystagmi.

Normally the horizontal "after-nystagmus" lasts from 15 to 30 seconds after 10 turnings; the rotatory a trifle longer; however, the duration of the horizontal after-nystagmus may be prolonged by the use of opaque spectacles or by having the patient look at a distant object to avoid convergence of the eyes which inhibits the nystagmus.

If, in a case of suspected right sided labyrinth suppuration the reaction to the right side is negative or of but a few seconds duration, while the reaction to the left side is 15 seconds or more, then the diagnosis is assured.

7. **Diminished or negative reaction to galvanism.**

Prior to six months ago the galvanic reaction of the labyrinth was a much neglected subject, owing to the crude and inaccurate methods used. Since then I devised a simple method of examination (Klinische Studien uber die Functions prufung des Labyrinthes mittlest des galvanischen Stromes. Archiv. fur Ohrenheilkunde. Bd. 77—1908), the examination is made with one pole in the hand and the second pole applied to the region of the ear, just in front of and above the tragus. The current is applied, gradually increasing until nystagmus is apparent and the strength of current necessary to produce the reaction noted in milliamperes. A record is made of the kathodal and anodal reaction of the two sides.

Normally 4 ma. with kathode to the right ear produces rotatory nystagmus to the right, and 4 ma. anode to the right ear produces rotatory nystagmus to the left, and vice versa for the left ear.

In a case of right sided labyrinth suppuration the kathode to the right ear fails to produce a reaction with as much as 8 ma. or more, while kathode to the left ear produces a reaction with less than 4 ma. In a similar manner we obtain the reactions with the anode (the right ear does not react).

In a similar manner we obtain the reaction with the anode (the right ear does not react).

A further examination of the opening and closing nystagmus of the two sides is as follows:

Right labyrinth acutely destroyed.
*R. Ear—K. O. N. > K. C. N.
A. C. N. > A. O. N.
L. Ear—K. C. N. > K. O. N.
A. O. N. > A. C. N.

The above reactions apply to the earlier stages of acute labyrinth suppuration; however, after secondary degeneration of the nerve has taken place it is impossible to obtain either kathodal or anodal reactions from the diseased side.

In closing the subject of diagnosis, I wish to emphasize that sudden deafness in the course of middle ear suppuration, together with spontaneous rotatory nystagmus to the sound side and vertigo, speak for labyrinth suppuration; but in addition, when the nonacoustic labyrinth of that side is nonreactive to the above mentioned tests, then the diagnosis of labyrinth suppuration is certain.

TREATMENT.

There is but one treatment for acute labyrinth suppuration; that is immediate radical operation combined with the labyrinth operation, for the following reasons:

1.—Labyrinth suppuration is frequently associated with intracranial complications; the more frequent of which complications are meningitis and cerebellar abscess.

2.—The prognosis in nonoperated cases is bad; the mortality being over 70 per cent., whereas the mortality from labyrinth operation is less than 8 per cent. While the mortality is high in the nonoperative cases, it is still higher in those cases where the radical operation is done and the labyrinth operation left undone than in those cases where no operation is done at all. In other words it is safer to leave a case of labyrinth suppuration to itself than to attempt any half way measures.

OPERATION.

The operation consists, first, in the regular Kuester-Bergmann radical; second, the laying free of the dura of the middle and posterior fossæ and removing the bridge of bone between them; third, removal

*K. O. N. = Kathodal Opening Nystagmus; K. C. N. = Kathodal Closing Nystagmus; A. O. N. = Anode Opening Nystagmus; A. C. N. = Anode Closing Nystagmus; > = greater than.

of the semicircular canals from behind and the free opening of the cochlea and vestibule in front, allowing the facial nerve to remain intact.

The dura is exposed for a double purpose:—

1.—To allow room for the labyrinth operation and

2.—To permit inspection of the membranes, since many of these cases have more or less meningitis as a complication.

The question of labyrinth suppuration is a very important one and almost too large for a single paper; my effort has been to cover as briefly as possible the entire subject.

LABYRINTH SUPPURATION—TWO CASES.*

THIS paper is intended to supplement a former paper upon "Diagnosis and Treatment of Labyrinth Suppuration."

The object is to report in full and discuss two cases of typical labyrinth suppuration. It is the opinion of the writer that a thorough report of one or at most two cases is worth more practically than a less thorough report of a greater number of cases.

The two cases here reported are selected from a series of twenty as the more classical cases of labyrinth suppuration. Copied from the records the cases are as follows:

Case I.—Franz G., 24 yrs. old, laborer.

Diagnosis.—Otitis Media Suppurativa Chronica Sinistra et Cholesteatoma et Labyrinthitis Suppurativa Acuta et Meningo-Encephalitis Serosa.

History.—Left ear has been discharging since early childhood. The patient could not recall exactly when or how it began. The discharge has not been continuous, often ceasing for months at a time. He had never been treated for the ear until ten days before admission. The left ear has been discharging continuously for the last seven weeks; during this time the patient complained of greater impairment of hearing in that ear and diffused headache, slight stiffness in the back of the neck and great weakness. Prior to this (seven weeks ago) the patient had never had vertigo, but at that time was taken with a very severe attack, which lasted several days. With the vertigo, he had the sensation as though he was being turned and complained of darkness before his eyes, he also vomited repeatedly with great nausea and he was compelled to lie down, which gave him slight relief. Upon questioning he had had no chill, and in his own words said he had had little or no fever. The patient came on account of discharge from and impairment of hearing in the left ear, intense headache, stiffness in the back of his neck and prostration.

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*The two cases here reported are taken from Prof. Alexander's clinic, of Vienna, with his permission. The examinations and records were made by the author when acting as Aspirant Assistant to Professor Alexander, but operated by him.

Present Condition.—Patient well nourished and muscular, sallow complexion and his facial expression suggests pain and apathy.

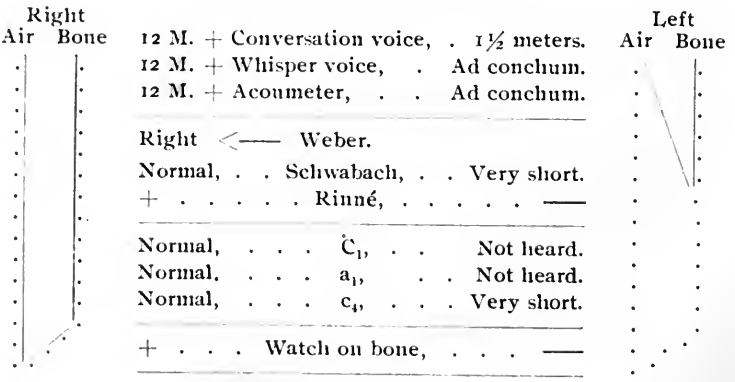
Otoscopic Examination—*Left Ear.*—Moderate amount of thick, grayish yellow, very fetid, purulent secretion in the external canal. Slight bulging of the superior wall of the canal; a mass of polyps about half the size of a pea hanging down from the attic-antrum region, which obstructed the view of the tympanic cavity, but it did not interfere with the introduction of the Hartmann canula for the purpose of washing out its contents, The mastoid process slightly sensitive to pressure. The entire left side of the head is more sensitive to pressure and percussion than the right side.

Right Ear.—Membrane slightly cloudy, otherwise normal.

Microscopic Examination of the more solid particles of the secretion obtained by syringing out the tympanic cavity of the left ear shows normal epidermis cells, epidermis cells in stages of degeneration, pus cells, fat crystals, *cholestearin* crystals, active cocci and bacilli and debris.

Ophthalmoscopic Examination showed marked choking of both discs (3 diopters).

Functional Examination.



SPONTANEOUS NYSTAGMUS.

Marked rotatory nystagmus to the right when looking to the right.

Very slight rotatory nystagmus to the right when looking straight ahead.

Slight horizontal nystagmus to the left when looking to the left.

DISTURBANCE OF EQUILIBRIUM.

Rhomberg positive. Hopping very uncertain and patient falls. Gait forward and backward with closed eyes very broad, uncertain and slow.

GONIOMETER.

Eyes Open.		Eyes Closed.
26 degrees,	Face forward,	20 degrees.
30 degrees,	Face backward,	22 degrees.
30 degrees,	Face to right side,	15 degrees.
30 degrees,	Face to left side,	15 degrees.

CALORIC NYSTAGMUS.

After three minutes' syringing of left ear with cold water the character of the spontaneous nystagmus was not changed (negative reaction).

Horizontal after-nystagmus to right, 18 seconds. Rotatory after-nystagmus to right, 15 seconds.	AFTER-TURNING NYSTAGMUS.	Horizontal after-nystagmus to left, 9 seconds. Rotatory after-nystagmus to left, 7 seconds.
Kathode, 3 ma. nystagmus to right increased. Anode, 7 ma., no reaction.	GALVANIC NYSTAGMUS.	Kathode, 7 ma., no reaction. Anode, 3 ma., rotatory nystagmus to right increased.

Negative, . COMPRESSION AND ASPIRATION NYSTAGMUS, . Negative.

THREE-METER HEARING TUBE. { Conversation voice, 50 per cent. failures.
Whisper voice, all failures.

Patient's temperature was 37° C., pulse 68.

Operation by Prof. G. Alexander.—Typical retro-auricular incision through soft tissues to bone. Laying free of the mastoid. Chiseling open the mastoid and antrum. The bone was sclerotic. The antrum and tympanic cavities were found filled with cholesteatoma masses, which were curetted out, together with many granulations. The radical operation after Kuester-Bergmann was carried out. Inspection of the horizontal semicircular canal showed a dirty, gray colored eroded area and a fistula (2 mm. broad and 5 mm. long). Two parallel horizontal incisions through the skin and soft tissues were made backward, 4 cm. long, from the upper and lower ends of the first incision. The mastoid process was totally removed and the posterior and middle skull fossæ freely exposed. Inspection of the sinus showed it to be normal. There

were no granulations on the dura. The semicircular canals were then removed from behind, the horizontal being left till last. Opening of the vestibule and accidentally the facial nerve was slightly exposed. The facial canal was eroded in part by the cholesteatoma. Chiseling open of the promontory. The entire labyrinth was softened. By incising the dura of the posterior and middle fossa a small amount of cloudy cerebrospinal fluid flowed out and the brain substance prolapsed through the incision. The dural incision was dressed with a quantity of iodoform gauze. Plastic was made after Panse. Wound dressing and bandage.

Lumbar puncture was then made, 15 cc. of cloudy fluid collected in three separate test tubes for further examination. The flow of fluid came with force indicating great pressure.

Immediately after the operation a slight facial paralysis in all branches of the 7th nerve was observed. The character of the nystagmus remained unchanged. The temperature 8 P. M. of the same day was 39 C. Patient was restless and an injection of morphine was given.

First day after operation there was no change in the facial paralysis. Less headache than before the operation. Temperature 37° C, pulse 68. Patient was able to sit up in bed and said he felt good.

Second day after operation, no headache, no vertigo, nystagmus unchanged. Patient had slept well and restful. Temperature 37.3° C and pulse 68.

Third day after operation, the patient felt generally good except for a slight frontal headache. Temperature 36.6° C, pulse 74. A purgative was given.

Fourth day.—First change of outer dressings.

Fifth day.—Patient slightly weak, otherwise the patient said he felt perfectly well.

Sixth day.—Second change of dressings. Wound showed healthy granulations.

For the next seven days the patient walked about the room at will and was free from symptoms.

Upon the fourteenth day after the operation a thorough functional examination of the nonacoustic labyrinth was made and the results were found to correspond exactly with the findings before the operation.

Fifteenth day, under local anesthesia with Schleich's solution, the retro-auricular wound was closed with secondary sutures excepting the lower tip for 1½ cm. Patient was discharged from the hospital with the advice to report every other day for further treatment.

One month after the operation the patient felt entirely well and had gained in weight. Retro-auricular wound entirely healed. Facial paralysis unchanged.

Six weeks after the operation the patient was free from all symptoms. Slight return of function of the 7th nerve. The nonacoustic labyrinth examination gave same results as before. There was still some slight mucoid secretion in the bottom of the canal. The patient was discharged and sent to his home, to be further treated by the local physician.

Case II.—Antonia K., 40 yrs. old, housekeeper.

Diagnosis.—Otitis Media Suppurativa Sinistra et Labyrinthitis Suppurativa et Meningitis Serosa.

History.—As a child began with discharge from the left ear, exactly as to how it began could not be ascertained. The discharge was intermittent, the longest intermission being five years. Between the second and twelfth years of age the patient suffered from tubercular glands in the neck and axilla. Also had repeated attacks of inflammation of the eyes (which proved to be keratitis eczematosa). Fifteen years ago the patient was operated for a left sided retro-auricular abscess (Wild's incision), after which the discharge ceased for five years. Six weeks before admisison to the hospital the patient had a heavy attack of vertigo, which lasted four or five days, during which time she vomited almost continuously and suffered greatly from nausea. She suffered greatly from thirst and thought she had fever, but everything she drank was vomited almost immediately. The patient was obliged to remain quietly in bed and every movement aggravated the vertigo. With the vertigo the patient complained of the sensation of the room and everything in it turning about her. Since then the vertigo has been gradually diminishing. Two weeks after this attack she went out of doors for the first time, had a slight chill and when she returned to the house she noticed that her face was crooked (facial palsy). Upon admission the patient complained of discharge and pains in left ear, impairment of hearing, slight vertigo when making quick movements, frontal headache—especially on the left side—and because of this last symptom the patient sought the hospital.

Present Condition.—Rather stout and well nourished woman. Scars in the neck from previous glandular involvement. Both eyes showed maculæ corneæ, but the fundi were negative.

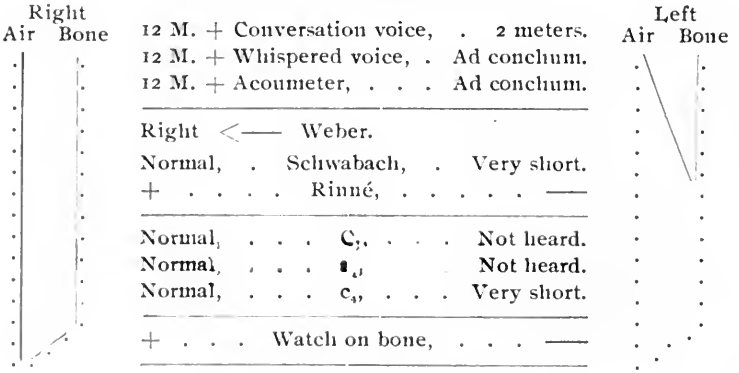
Otoscopic Examination:

Left Ear.—Moderate amount of brown, very fetid, purulent secretion, external canal slightly narrowed. Large polyps hanging down from the attic-antrum region which make it impossible to see the tympanitic cavity. The mastoid process is slightly sensitive to pressure, the periosteum thickened and adherent to the bone. There is an old, one cm. long, vertical scar behind the left auricle representing the Wild's incision which had been performed several years before.

Right Ear normal.

Microscopic Examination of secretion from left ear showed a few normal and degenerated epidermis cells, many pus cells, movable cocci and bacilli, a few fat crystals and debris; but no cholesterin crystals.

Functional Examination.



SPONTANEOUS NYSTAGMUS.

Marked rotatory nystagmus to the right when looking to the right.

Slight rotatory nystagmus to the right with opaque glasses when looking straight ahead.

Slightly rotatory combined with horizontal nystagmus to the left when looking to the left.

DISTURBANCE OF EQUILIBRIUM.

Rhomberg positive but slight. Gait forward and backward with closed eyes somewhat broad and very uncertain. Patient tends to fall laterally, but not to either side particularly. Hopping impossible.

GONIOMETER.

Eyes Open.		Eyes Closed.
24 degrees,	Face forward,	7 degrees.
22 degrees,	Face backward,	7 degrees.
23 degrees,	Face to right side,	3½ degrees.
23 degrees,	Face to left side,	3 degrees.

CALORIC NYSTAGMUS. Negative.

Horizontal after-nystagmus to right, 30 seconds. Rotatory after-nystagmus to right, 14 seconds.	AFTER-TURNING NYSTAGMUS.	Horizontal after-nystagmus to left, 9 seconds. Rotatory after-nystagmus to left, 3 seconds.
Kathode, 2 ma., nystagmus to right increased. Anode, 12 ma., nystagmus to left.	GALVANIC NYSTAGMUS.	Kathode, 12 ma., nystagmus to left. Anode, 7 ma., nystagmus to right.

COMPRESSION AND ASPIRATION NYSTAGMUS. Negative

THREE-METER HEARING TUBE. { Conversation voice, 70 per cent. failures.
Whispered voice, all failures.

Operation by Prof. G. Alexander.—Typical 6 cm. long retro-auricular incision to the bone. Exposure of the mastoid in front of and behind the incision. Separation of the membranous canal from the posterior, superior and inferior bony canal. Opening of the mastoid and antrum. Upon opening the antrum pus flowed freely. Removal of the posterior bony canal with bone forceps, carrying out of the radical operation after Kuester-Bergmann. The middle ear spaces were thoroughly curetted of granulations and pus. Curettement of the Eustachian tube. Laying free the dura of the posterior and middle fossæ and the sinus wall. A granulation the size of a pea was found on the sinus wall. A fistula was uncovered, which led to the lower part of the superior semicircular canal from behind, opening of vestibule, opening of the promontory in front of the facial canal so that a bent probe passed through from behind the facial canal could be seen anteriorly. Clear liquid oozed through the anterior opening. Free incision of the dura of both fossæ through which edematous brain substance prolapsed. Plastic after Panse. Abundant dressings of iodoform gauze were applied over the dural incisions, over this sterile gauze and bandage.

Several attempts at lumbar puncture were unsuccessful, owing probably to excessive amount of adipose tissue in this region.

Evening of the same day the patient complained of some headache,

slight vertigo and had vomited two or three times. Temperature 36.8° C., pulse 100.

First day after the operation the patient complained of headache, partial facial paralysis of all branches, slight evidence of amnestic aphasia and patient also complained of being confused.

Second day: Headache same as yesterday; patient had ceased vomiting. Writing test showed that she omitted letters in words and she complained of being forgetful. Examination of fundi again negative. Outer bandage was slightly loosened.

Third day: Patient still complained of headache and could not sleep. Dressings were changed, showing a moderate amount of fetid secretion.

Fourth day: Headache is less, facial paralysis less. Eye grounds again examined and found negative. Patient felt fairly well.

Fifth day: Headache and paralysis better. Temperature and pulse normal. Second change of dressings and wound showed normal reaction.

Seventh day: Third dressing; patient felt so well that she was allowed to leave the hospital to report every other day for further treatment.

End of fourth week. Patient felt well, was doing her regular housework. At this time a thorough functional examination was made which gave the same results as before the operation (see above).

End of two months the patient was discharged cured.

DISCUSSION OF THE CASES.

The taking of the history is very important, especially in cases of labyrinth suppuration. In taking the history I have adopted a plan first suggested by Alexander, *i. e.*,

1.—When did the suppuration of the middle ear begin?

2.—How did it begin? Suddenly, with pain, with fever, how long did these acute symptoms last before the discharge began or did it begin gradually without acute symptoms?

3.—Has the discharge been continuous or periodic?

4.—Has the ear been treated?

5.—Of what does the patient complain? Discharge, impairment of hearing, subjective noises, vertigo and its character, fever, headache, and questioning for further symptoms according to what may have suggested itself up to this time.

6.—Finally a very brief summary of symptoms most complained of by the patient. This can be best accomplished by putting to the patient the question—for what do you come?

Observing the histories of the cases reported the reader will notice that this method of history taking has been adhered to.

We shall now discuss the histories of these cases.

(a) Both cases began in early childhood; which shows that they were chronic suppurations of the middle ear.

(b) The character of the onset was not ascertainable, since the patients could not recall it, and no known cause in either case.

(c) The discharge was periodic in both cases. This should prove to us the importance of keeping under observation all cases of apparently healed chronic middle ear suppuration for a long time after cessation of discharges from the ear; furthermore, the verbal report of the patient "that he has no further discharge from the ear" should not be accepted, since in many cases the discharge may be so slight as to escape the notice of the patient.

(d) In both cases the treatment had been more or less neglected. In one case there had been no treatment until ten days before admission to the hospital. In the other case only partial and improper treatment, including a Wild's incision performed several years before;—a treatment long abandoned and condemned by the best operators.

(e) Both complained of the following symptoms: Otorrhoea, marked impairment of hearing,* vertigo, slight fever (?) and headache.

Neither case complained of subjective noises. Relative to this symptom: I have never found subjective noises in a case of labyrinth suppuration—as some of the American writers have claimed; however, subjective noises may be present occasionally just before the onset, but never after the actual development of the labyrinth suppuration, and indeed there seems to be no logical reason why there should be subjective noises in a case of suppurative panotitis any more than there should be subjective sensations of light in a case of suppurative panophthalmitis; however, this is only a minor point which plays no important part in the diagnosis.

As to the vertigo, it was slight at the time of the examination but had been very intense several weeks previous, lasting several days and gradually diminishing. The memory of the vertigo was vivid enough in both instances for the patients to describe it accurately. One patient described it as a sensation of apparent turning of the surrounding objects; the other, a sensation of apparent turning of himself.† Both patients had marked nausea and vomiting as accompanying symptoms, and were compelled to lie down in order to feel

*Actual deafness is rarely complained of in any case having from good to perfect hearing in the remaining ear. The determination of complete deafness must be decided by the functional tests.

†The patient who experienced the sensation of surrounding objects turning was more observing and less neurotic than the patient who experienced only the sensation of being turned himself. Darkness before the eyes is a late symptom of vertigo found especially in neurotic people.

secure from falling and to diminish the intensity of the vertigo. Notwithstanding the fact that the patients described their sensations somewhat differently, the description of one is as characteristic as that of the other for vestibular vertigo (*Drehschwindel* of the German authors).

Vestibular vertigo then is our first cardinal symptom of labyrinth suppuration, elicited from the history of these cases.

Fever was difficult to determine and if it had been present must have been very slight, since the one patient who believed she had fever decided so from the thirst she suffered. Thirst is a factor in determining fever only with the laity and in this case was due more to the excessive vomiting than to anything else. The other patient was inclined to the belief that he had had no fever.

Headache was pronounced in both cases. I have come to regard headache, though not a cardinal symptom, yet one of importance in the differential diagnosis between suppurative and serous labyrinthitis. The reason is very apparent; since in the serous labyrinthitis the membranes of the brain are found to be normal while in the suppurative form I have frequently found in early cases serous meningitis. These two cases were no exception to this rule. In addition, Case I. complained of apathy and stiffness behind the neck. Case II. complained of a crooked face with inability to close the left eye, indicating 7th nerve palsy.

Summary of the history in both cases showed in common: Chronic middle ear suppuration of unknown origin, intermittent discharge from the ear, neglected treatment, impaired hearing, typical vestibular vertigo and symptoms of headache indicating some form of meningitis or meningo-encephalitis.

From the histories alone I was able to make a *provisional* diagnosis of labyrinth suppuration, fixing the date of the attack, and that secondary meningitis was present at the time.

Otoscopic findings.

Both cases presented a moderate amount of dirty looking, fetid, purulent discharge from the left ear. The fetidity of the discharge in these cases was not so important as in other cases where the ear had been recently treated. (Persistent fetidity of the discharge after thorough and repeated cleanings should lead one to suspect cholesteatoma).

The external bony canal was narrowed in both cases, indicating recent mastoiditis. Mastoiditis in these cases was more coincidental than characteristic for labyrinth complications since I have found as many cases free from mastoiditis as with it.

Polyps were found in both cases hanging downward from the attic-antrum region into the mesotympanum which prevented a thorough inspection of the tympanic cavity. I did not remove these polyps in order to make a more thorough inspection of the tympanic cavity for

the following reason: It is a safe rule *never to do a minor intra-tympanic operation* (polyp extraction or ossiculectomy) *when a radical operation is otherwise indicated.*

Tenderness over the mastoid was more or less present in both cases and in one case the periosteum was thickened.

Microscopic examination of the syringed out tympanic secretion, examined in the natural state with a 1/8 objective, showed epidermis, pus cells, movable micro-organisms, debris, and in addition Case I showed the typical cholestearin crystals, indicating cholesteatoma, which was later corroborated at the operation.

For a more detailed description of cholesteatoma and methods of diagnosis, the reader is referred to a paper upon "Cholesteatoma" by the, writer, Monatsch. f. Ohren., 1908. Zur klinischen Diagnostik des Mittelohrcholesteatoms.

Eye ground was normal in Case II, and choked discs found in both eyes in Case I.

Excepting for the ear and head conditions the general condition of the patients was fair.

Functional examination of the ears showed both acoustic and non-acoustic functions to be negative in the diseased (left) ear of both patients.

Conversational voice was heard from 1½ to 2 meters upon the left side and whispered voice and acumeter ad conchum (less than 20 cm.). This amount of hearing from the left side does not prove that the patients actually heard with the left ear. Bezold, Politzer, and recently Alexander, Barany and others have shown, in cases of one sided deafness, that exclusion of the normal ear by the ordinary means of stopping up the canal with a wet finger or with paraffine is insufficient. Repeated experiments with cases of absolute one sided deafness (cases where the labyrinth of that side had been removed) show apparent hearing of from 1 to 6 meters distance for conversational voice. It was therefore necessary to try other methods, the 3 meter speaking tube recommended by Politzer and Bezold's new small a_1 fork, manufactured by Edelman. The telephone method and the new method of Barany were not tried.

When the speaking tube shows more than 50 % failures to conversational voice and 100 % failures to whispered voice, the Politzer school accepts such cases as those of complete deafness. I have, however, greater faith in the Bezold small a_1 fork.

I make the examination by striking the fork a moderate blow with a rubber-covered hammer so that the fork can be heard distinctly at 7 or 8 inches, the width of the skull, but no further. When the fork thus struck is held 1 inch from the normal ear it can be heard very intensely. I have found this method of examination the best for producing loud tones when held close to the ear and at the same time excluding all possibility of hearing from the opposite ear. I can recommend this fork as the best asset an otologist can have for making functional hearing tests for one sided deafness.

The low C_1 was not heard, and the high c_4 was heard but very much shortened. These high tone forks are always unreliable tests for one sided deafness since they can be heard at very long distances and have great power of penetration.

The remaining tests—negative Rinné, with the middle C with no air conduction and very much shortened bone conduction; Weber to the better ear;* Schwabach very short; negative watch on bone are the same results as obtained in every case I have examined.

From the above functional test findings we must conclude that both patients were absolutely deaf on the left side; this, therefore, makes a second positive sign or symptom of labyrinth suppuration—*Absolute deafness.*

EXAMINATION OF THE NONACOUSTIC LABYRINTH.

Both cases showed pronounced spontaneous rotatory nystagmus to the sound (right) side upon the patients looking to the right; less marked horizontal and mixed horizontal and rotatory nystagmus to the left side upon looking to the left, and slight but evident spontaneous rotatory nystagmus to the right side when looking straight ahead; however, in determining this last form of nystagmus it was necessary in one of the cases to use the opaque spectacles.

The above findings are characteristic of late labyrinth suppuration after the first, second or third week. In acute cases, however, the nystagmus is constantly rotatory and to the sound side in all positions of the eyeballs. Most marked when looking to the sound side, less marked when looking straight ahead and least marked when looking to the diseased side. This nystagmus gradually diminishes, and after a few weeks changes to the character as recorded above and presented by the cases here reported.

The rotatory nystagmus to the sound side when looking straight ahead I consider the most important and characteristic spontaneous nystagmus of *late* labyrinth suppuration.

In making the examination for spontaneous nystagmus in the straight ahead position of the eyeballs, it is necessary first to have the patient look at a distant object or else use the opaque spectacles to prevent convergence, which inhibits the vestibular reflex; second, elevate the lids sufficiently (preferably with the thumb of the left hand when examining the right eye and vice versa when examining the left eye) to observe a fair portion of the sclera above the cornea (see Fig. 1); third, a good light thrown upon the eyeball which helps us to see

*Neumann, of Vienna, has told to his classes that he had observed in at least two cases of one sided labyrinth destruction, Weber lateralized to the diseased side. These were cases of total sequestration of the labyrinth. He believes that in cases of one sided labyrinth destruction, secondary to middle ear disease, with complete loss of acoustic and static functions, that Weber to the diseased side indicates a perilabyrinthitis.

well and at the same time makes a reflex upon the eye which gives us a stationary point by which we can estimate the movements of the eyeball by the change of relationship between the reflex and one of the prominent vessels of the sclera.

The intensity of vestibular nystagmus is proportionate to the frequency of the movements and the length of the excursions. From this we can appreciate the ease of determining the character and di-

FIG. 1.

Illustrates a method of examination of nystagmus of slight degree. Since the voluntary action of the III nerve in accommodation and convergence inhibits the vestibular reflex through the Deiter's system, Abels suggested the use of the opaque spectacles. The spectacles are used also in making more accurate examinations of the horizontal after-turning nystagmus. The spectacles are fitted upon the patient as the ordinary spectacles are, the patient is directed to look straight ahead into vacancy and with the head slightly tilted backward, the thumb used to elevate the upper lid, we observe the nystagmic movements of eyes over the top and side of the spectacles.

rection of the nystagmus in an acute case and the difficulty of determining the nystagmus in a late case. However, exercise in examining a number of cases will make one quite proficient after a short time. In the cases here reported the length of the excursions was approximately 2 mm. and the frequency about 5 seconds; a casual glance would not suffice to recognize it. This type of nystagmus

is characteristic of late labyrinth suppuration. Thus we have our third cardinal symptom or sign—*Spontaneous rotatory nystagmus to the sound side.*

Disturbances of equilibrium belonging to the late form of labyrinth suppuration were present.

It will be remembered from the former paper that I referred to an early and a late form of disturbance of equilibrium. The case here reported could naturally show only the late form; which was evidenced by a slight but positive Rhomberg:* broad and uncertain gait with closed eyes when walking forward and backward; unsteadiness was very pronounced by side gait and hopping on one foot with closed eyes. The Alexander-Stein goniometer showed diminished ability to maintain equilibrium. According to Stein, a normal person can tolerate an elevation on the inclined plane of from 30° to 34° when the face is forward, backward or to the sides and with closed eyes,† but in these cases the figures fell much below the normal; thus, we have a fourth symptom of labyrinth suppuration—*Disturbance of equilibrium.*

The examination of the nonacoustic labyrinth was made after three methods—1, caloric; 2, after-turning; 3, galvanic.

1. The **caloric examination** elicited no reaction from the diseased ear in either case.

In making the caloric examination I use a Hartman's canula fitted on a rubber tube about 25 cm. long, to the other end of which is attached a 10 oz. Politzer bag filled with cold water (about 20° C). I prefer the use of cold water, since the reaction when present is more prompt and more certain than when hot water is used. The application of cold water is best made by an assistant and the observation of the reaction (nystagmus to the opposite side) by the surgeon. After first squeezing out the air, the tip of the canula is introduced through the external canal into the tympanic cavity. A good light and a speculum are essential aids for the successful introduction of the canula.

The observer's position is in front of the patient with a head mirror directing the illumination upon the eye; with the thumb of the left hand the upper eyelid is raised to better expose the eyeball (see Fig. 2). At the moment of appearance of reaction, stop the stream of cold water and begin to neutralize the effect with hot water (42° C.). The neutralizing is done with hot water to cut short any unpleasant symptoms of vertigo and vomiting, which otherwise frequently continue for some time. It is worth mentioning that the nystagmus precedes the vertigo by a few seconds and that low degrees of nystagmus can be ob-

*Rhomberg test is the least accurate of all equilibrium tests.

†I make all tests with patients standing in bare feet, since the shoes in some instances are an aid, but more often a hindrance, in the effort of the patient to maintain equilibrium.

tained without much vertigo. The above reaction applies only when making examinations with the head in the upright position.

This method of examination is satisfactory enough in late cases of labyrinth suppuration where the nystagmus is slight; but not in a recent case, since a recent labyrinth suppuration has pronounced rotatory nystagmus to the sound side and the reaction from the use

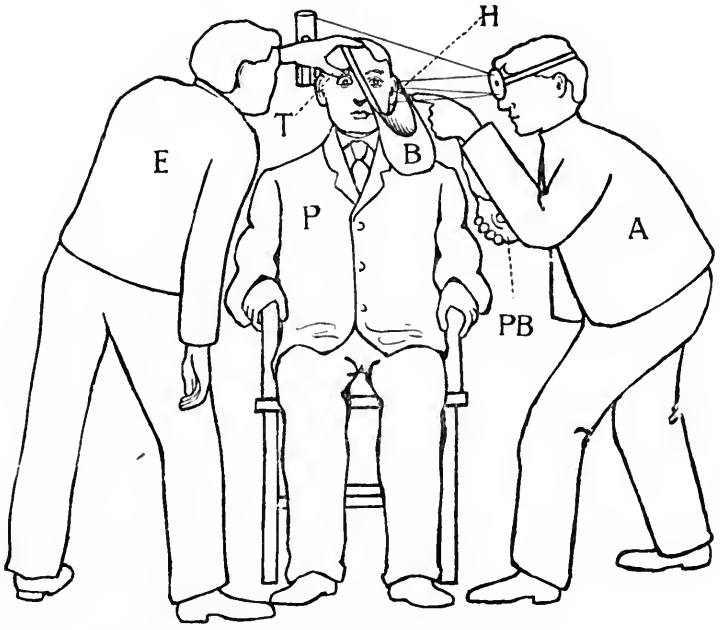


FIG. 2.

P, Patient is looking straight ahead at some distant object, as directed; A, assistant syringing ear using the Hartmann's canula, H, and P B, Politzer bag filled with water to be syringed. B, pus bag to collect the waste water and discharges from the ear. E, examiner raising the upper eyelid with thumb, T, to better observe the nystagmic movements of the eyes.

This method can be improved upon if one uses a fountain syringe, then the left hand can be used to hold speculum in place, the right hand to direct the canula.

of cold water could only increase the nystagmus; this allows a chance for an error of observation in determining a positive reaction. It is therefore more satisfactory in such acute cases with pronounced rotatory nystagmus to the sound side to make the caloric reaction with cold water as described, but with the head in the horizontal position;

for example—the right side of the face upon the right shoulder when examining the left ear, when a positive reaction is indicated by a horizontal nystagmus to the diseased side (left).

In the case here reported no caloric reaction was obtainable after 3 minutes' continuous flow of cold water in the tympanic cavity; thus, we have our fifth sign of labyrinth suppuration—*Negative caloric reaction.*

2. **Diminished or Negative reaction to turning.**

Diminished or negative nystagmus after turning would be a more corect way of expressing the reaction and hereafter I shall adopt the term "After-nystagmus" which is the literal translation of "Nach-nystagmus" of the German literature (Barany). Another reason for abandonment of the former term is that when speaking of diminished reaction to turning one may be misunderstood to mean that in labyrinth suppuration there is, though diminished, a positive reaction from the diseased labyrinth, whereas the fact is there is no reaction from the diseased labyrinth and the nystagmus which is present toward the diseased side (after turning to the well side) is due to the normal physiological reaction of the opposite (well) side.

While upon the subject a brief explanation is opportune. Ewald was the first to prove the physiology of the semicircular canals by means of a pneumatic hammer fitted into an opening made in the bony horizontal semicircular canal of a pigeon, permitting the membranous labyrinth to remain intact; he was able to cause a flow in the endolymph from the smooth end toward the ampulla by pressure, and, from the utriculus and ampulla toward the smooth end by suction. He found furthermore that the flow of the endolymph from the smooth end towards the ampulla caused a movement of the cupola and an inclination of the cilia of the neuroepithelia in the ampulla towards the utriculus and with it a horizontal nystagmus of considerable intensity towards the same side; while a flow of the endolymph in the opposite direction caused the opposite effect (a horizontal nystagmus of less intensity towards the opposite side). In brief, the semicircular canals of one side are capable of producing nystagmus to both sides, depending upon the direction of the endolymph current, but stronger to the same than to the opposite side; so that in case of total destruction of one side we can obtain a nystagmus to the diseased side, but much diminished in intensity and duration.

Since the semicircular canals of one side can produce nystagmus to either side but more pronounced to the same than to the opposite side, and since by rotation of the head both labyrinths are brought into function, it must follow that in a case of left labyrinth suppuration (destruction), the nystagmus to the left side is greatly diminished and the nystagmus to the right side but slightly. These facts are furthermore borne out by the experience of all who have investigated after-turning nystagmus.

The examination is made with the patient sitting upon the revolving stool as seen in Fig. 3.

The patient sits in the chair while the examiner revolves it 10 times,* then suddenly brings it to a stop and, with a stopwatch times the duration of the after-nystagmus. The examination for horizontal after-nystagmus should be made by turning the patient with the head in the upright position, while the examination for rotatory after-nystag-

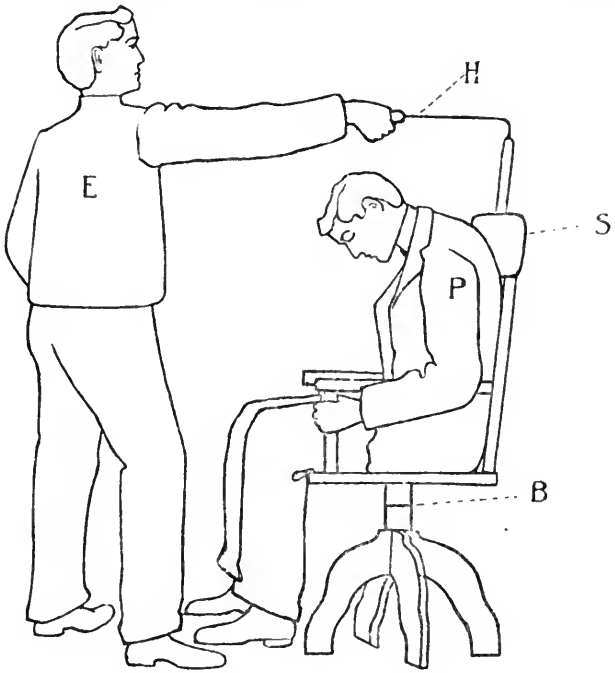


FIG. 3.

P, patient; E, examiner; S, revolving stool; B, bearing, substituted for the ordinary screw, permitting rotation of the stool without the elevation or lowering which occurs when the stool is fitted with a screw. H, handle. In the figure the patient is in a position (head forward) to produce rotatory nystagmus by turning or after stopping turning. The eyes are closed to avoid optical nystagmus.

mus should be made with the head inclined forward 90°, as in Fig. 3; however, the *observation* of the rotatory after-nystagmus is made

*Turning ten times has been largely adopted instead of a greater or less number of times for evident reasons. See Barany, "Physiologie und Pathologie, des Bogengang Apparates beim Menchen."

with the head upright. In other words, we may change the position of the head from the forward to the upright position after turning without changing the character of the nystagmus.

The duration of the after-nystagmus varies greatly; in normal people the average being 26 seconds for rotatory and 35 seconds for the horizontal. A diminution to 12 seconds for either horizontal or ro-

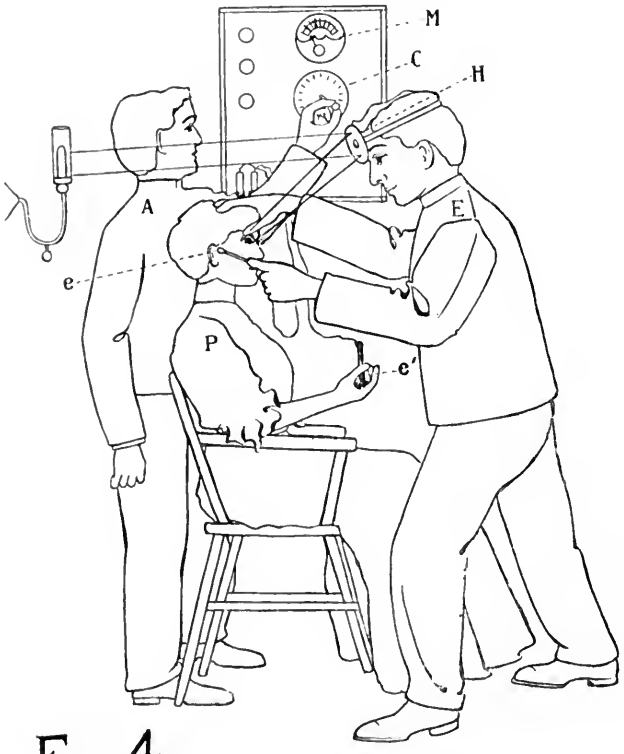


FIG. 4

P, patient; A, assistant who runs the switch, C, and reads the milliamperage from the milliamperemeter M; E, examiner; H, head mirror to illuminate the eye; e and e', electrodes. The wall plate should show a reversing switch to change the polarity without changing the electrodes.

tatory after-nystagmus is an indication of destroyed function upon that side, and according to some a diminution to 15 seconds is sufficient for a diagnosis. I am more conservative, however, since I found two cases—one with 14 seconds and one with 13 seconds' duration—where the labyrinths were still in function.

In the cases before us the longest duration for the after-nystagmus

to the diseased side was 9 seconds in one of the cases for the horizontal nystagmus with the opaque spectacles. Such low figures are never found in a reacting labyrinth, and with even lower figures for the rotatory after-nystagmus clinches the fact that the nonacoustic labyrinths upon the diseased side were destroyed. Thus, the sixth symptom or sign of labyrinth suppuration present in these cases—*Diminished after-nystagmus to the diseased side.*

3. **The Galvanic reaction** in both cases shows a characteristic diminution of irritability upon the diseased side. Normally from 4 to 6 ma. applied with a small ball electrode 1 cm. in diameter just in front of and above the tragus (see Fig. 4) will produce a nystagmus (with vertigo) to the side of the kathode when the kathode is used, and to the opposite side when the anode is used. A reaction with less than 4 ma. with the kathode indicates over-irritability of that side or destruction of the opposite side. A reaction which requires more than 8 ma. indicates loss of function in the static labyrinth of that side or over-irritability of the opposite side.

The question which would naturally suggest itself to the average reader is—Why should we conclude that there is destruction in the labyrinth when we can obtain a positive reaction with 8 ma. with the kathode to the diseased side? The answer is that in such cases the positive reaction is obtained from the nerve and not from the end organ in the semicircular canals.

The Galvanic reaction is so large a subject that a thorough discussion of it would take up too much space for this paper. I shall therefore reserve for another paper upon "Differential Diagnosis" a thorough discussion of the Galvanic reaction, where it plays a more important role.

In brief, the characteristic reaction for labyrinth destruction of not too long standing is:

Left Ear (labyrinth suppuration).—Kathode, 8 to 10 ma., no reaction or slightly positive rotatory nystagmus to left side. Anode, 2 ma., rotatory nystagmus to right side.

Right Ear (normal).—Kathode, 2 ma., rotatory nystagmus to right side. Anode, 8 ma., no reaction or slightly positive rotatory nystagmus to the left side.

Again, if the kathodal reaction to the diseased side requires more than twice the number of milliamperes as that to the normal side we may conclude that there is loss of function upon the diseased side.

The Galvanic reaction in the cases presented is characteristic for the labyrinth suppuration; hence our last positive symptom or sign of labyrinth suppuration—*Diminished* or *negative Galvanic reaction upon the diseased side.*

The operation, the subsequent treatment and results require no special discussion and are sufficiently clear to speak for themselves.

SUMMARY.

I. All symptoms and signs were positive for the diagnosis of labyrinth suppuration.

II. Symptoms and signs were present sufficiently characteristic to warrant a diagnosis of meningitis and exposure and examination of the membranes at the time of operation.

III. The operations consisted of the radical Kuester-Bergmann plus thorough opening of the labyrinth and the operation for serous meningitis recommended by Koerner and Alexander.

IV. With the exception of facial palsy, which is rapidly improving, both cases experienced early improvement of meningeal symptoms after operation and ultimately were discharged cured.

LABYRINTH SUPPURATION—DIFFERENTIAL DIAGNOSIS.

WHILE the otologists are paying increased attention to the subject of labyrinth suppuration it is well that we pay even greater attention to the subject of differential diagnosis, for with the advances made in its surgical treatment there is the danger that some of us, in our enthusiasm to operate, may sacrifice unnecessarily labyrinths of patients (presenting the history of chronic suppuration and the symptoms of vertigo, nystagmus and disturbance of equilibrium) who would otherwise recover spontaneously.

It is an appreciation of these dangers which prompts the writer in the presentation of this paper upon Differential Diagnosis. Of all the cases presenting the above history and symptoms only a relatively small per cent. are suffering from labyrinth suppuration.

We shall consider only those affections of the labyrinth secondary to acute or chronic middle ear suppuration which, by reason of the symptoms of impaired hearing, vertigo, nystagmus and disturbance of equilibrium, may be confounded with labyrinth suppuration. They may be divided into:

I.—Affections of the membranous labyrinth:

A.—Diffuse
1.—Diffuse hyperemia.
2.—Diffuse serous labyrinthitis.
3.—Diffuse suppurative labyrinthitis (labyrinth suppuration).

B.—Circumscribed
1.—Circumscribed irritative lesion of the membranous labyrinth.
2.—Circumscribed destructive lesion of the membranous labyrinth.

II.—Affections of the bony labyrinth (erosions, caries, necrosis, fistulæ, etc.).

III.—Affections of the perilabyrinthine spaces (perilabyrinthitis with resulting sequestration of a part or the entire labyrinth.

I, A, 1.—DIFFUSE HYPEREMIA OF THE LABYRINTH may be met with in the course of acute or chronic middle ear suppuration. The opinion

of some authors is that every acute middle ear inflammation is attended with more or less hyperemia of the labyrinth, while others believe that the labyrinth symptoms are due rather to the pressure of confined secretion upon the windows; it would seem that both views are in a measure correct. In support of the first view we know of cases of acute infections of the ear where the greater force of the infection has been felt in the labyrinth (especially infection from streptococcus mucosa) with resulting labyrinth suppuration and meningitis, whereas the middle ear affection remained so mild that it did not lead to even perforation of the membrane; while on the other hand, in support of the second view, there are cases of severe acute otitis media with associated labyrinthine symptoms which are promptly relieved by paracentesis of the membrane.

Aside from the cases of panotitis which terminate fatally within a few days after the onset of acute otitis media, the opinion of the writer is that every acute middle ear inflammation is accompanied by more or less congestion of the labyrinth; its intensity varying in proportion to the virulence of the infecting micro-organism.

Hyperemia of the labyrinth may occur too in the course of chronic middle ear suppuration, especially during periods of acute exacerbations. The cause in this instance is not exactly known. It is probably due to toxins generated by the infection in the middle ear which find their way into the labyrinth.

Hyperemia and the symptoms of hyperemia may begin suddenly like those of labyrinth suppuration, in spite of which and the fact that they have several symptoms in common, the two processes are very unlike; for in hyperemia we have an irritative process with increased reactibility; while in suppuration we have a destructive process with negative reactibility.

A comparison of the two processes shows the following:—

HYPERÆMIA OF THE LABYRINTH.	SUPPURATION OF THE LABYRINTH
1.—Impairment of hearing increased.	1.—Absolute deafness.
2.—Tinnitis.	2.—*No* tinnitis.
3.—Moderate vertigo, *aggravated* in recumbent position.	3.—Intense vertigo, but constantly diminishing and *relieved* in recumbent position.

4.—Spontaneous nystagmus to *both sides,* but more marked to the the *diseased* side.	4.—In acute stage nystagmus to the *well* side only; later stages it may be to both sides, but always more marked to the *well* side.
5.—Irritability of the labyrinth *increased.*	5.—Irritability of the labyrinth *negative.*
6.—Disturbance of equilibrium not so pronounced as in suppuration of the labyrinth.	6.—Disturbance of equilibrium very pronounced in the acute stage; present, though moderate, in the last stage.
7.—Ultimate *recovery* of acoustic and static functions.	7.—Permanent *loss* of acoustic and static functions.

The increased reactibility of the labyrinth in hyperemia is determined by the turning and galvanic test (quantitative tests). Since the caloric test can be only positive or negative, it becomes purely a qualitative test, and can not be used as a quantitative one.

The increased reactibility of the labyrinth is manifested by an increase in the duration of the *after-nystagmus* to the diseased side; for example—if the right labyrinth is over irritable we have an *after-nystagmus* to the right lasting from 40 to 100 seconds or more, while the *after-nystagmus* to the left remains about the normal figures, say from 18 to 35 seconds. It may also happen in cases where one labyrinth is very markedly pathologically irritated, that the *after-nystagmus* to the well side is also relatively increased.

The galvanic reaction in case of hyperemia, say of the right labyrinth, shows that the right labyrinth with kathode to the right ear responds to irritation with less milliamperage than the opposite (well) ear. In addition to the above signs I have frequently found in cases of hyperemia of the labyrinth associated with acute middle ear inflammation, a marked compression and aspiration nystagmus, a symptom *never* found in suppuration of the labyrinth.

This compression and aspiration nystagmus in acute cases may be found before perforation, or after when the perforation is very small. I believe it is due to the hydraulic pressure of the secretion in the middle ear upon the windows being greater than the pneumatic pressure when the middle ear is in the normal state. Furthermore, in the acute inflammatory conditions the pressure of the secretion, by reason of the

closure of the Eustachian tube by inflammatory swelling, is confined more to the walls of the tympanic cavity, while in the normal state the pressure is not so confined to the middle ear but escapes appreciably through the patulous Eustachian tube.

The differential diagnosis of serous from suppurative labyrinthitis may be quite difficult.

I, A, 2.—SEROUS LABYRINTHITIS.

Serous Labyrinthitis is a serous inflammation of the membranous labyrinth produced by the presence of toxins, while suppurative labyrinthitis is due to the infecting micro-organisms themselves. In serous labyrinthitis we have a serous or a mildly serofibrinous exudate into the labyrinth, which later may become partially or completely resorbed with corresponding restoration of function. In suppurative labyrinthitis we have a violent inflammation of the labyrinth with the formation of pus, resulting in total and permanent destruction of the membranous labyrinth with loss of function.

The prognosis in serous labyrinthitis is generally favorable; the result being a partial or complete restoration of function; notwithstanding this, a serous labyrinthitis is often the precursor of a suppurative labyrinthitis, a fact we should reckon with in considering the treatment. At their height the two processes are very similar, the symptoms and signs are perhaps not quite so pronounced in the serous as in the suppurative form.

A sign of importance in differential diagnosis which I have noted is the headache, of meningeal type, present in suppurative labyrinthitis, which is absent in the serous. This headache is due to irritation or serous inflammation of the meninges, termed by some few writers as meningismus, but accepted by most as a serous meningitis. This headache appears very early, often obscured somewhat at the beginning by the intense vertigo. Aside from the headache and the difference in the courses of the two processes we have nothing else to aid us in the differential diagnosis. These facts can be best illustrated by the comparison of the two tables below, showing the symptoms and the course of the two processes:

DIFFUSE SUPPURATIVE LABYRINTHITIS.

STAGES	I	II	III	IV	V
	Normal condition before labyrinth involvement.	Prodromal or irritative stage; may last from few weeks to several years.	Onset of suppuration sudden.	From several days to several weeks after onset.	End stage; destruction of functions.
Hearing.	+	+ But diminished.	—	—	—
Tinnitus	—	+ Periodic.	—	—	—
Vertigo.	—	+ In attacks	+ Intense	+ Only during rapid movement of head.	—
Spontaneous Nystagmus.	—	To both sides; more marked to diseased side	Intense to well side only.	Both sides; more intense to well side.	—
Irritability of the static labyrinth.	Normal	+ and increased.	—	—	—

DIFFUSE SEROUS LABYRINTHITIS.

STAGES	I	II	III	IV	V
	Normal condition before labyrinth involvement.	Prodromal or irritative stage.	Onset of serous labyrinthitis.	Stage of improvement.	Recovery
Hearing.	+	+ But diminished.	—	+ But diminished.	+

DIFFUSE SEROUS LABYRINTHITIS.—(Continued.)

STAGES	I	II	III	IV	V
Tinnitus.	—	+ Periodic.	—	+	—
Vertigo.	—	+ In attacks.	+ Intense	+ Only by rapid movement of the head.	—
Spontaneous Nystagmus.	—	To both sides; more marked to diseased side	Intense to well side only.	To both sides; more marked to diseased side	—
Irritability of the static labyrinth.	Normal.	+ and increased	—	+ with caloric test.	Normal or nearly so.

The above tables represent fairly accurately the course of these two processes. I have presented stage I as the normal condition; stage II the stage of iritation and of prodromes. Although both serous and suppurative labyrinthitis have a sudden onset, they are preceded by a longer or shorter period of irritation with attacks of vertigo and nystagmus to the well side. III is the stage of attack with sudden onset, accompanied by deafness, intense vertigo and nystagmus to the well side and negative reaction of the static labyrinth. These stages are more or less sharply defined, and up to and including stage III the two conditions are clinically alike with the exception of headache in the suppurative form, already mentioned above. After stage III the two processes begin to differ and eventually in stage V we have the end stage of suppuration, complete loss of acoustic and static functions, while stage V of serous labyrinthitis records the normal findings or those existent before the attack, as in stage I.

The differentiation of these two forms of labyrinthitis is quite difficult until stage III has passed, and where there is doubt it may be well to wait a few days or a week after the onset before deciding upon the treatment. If there is much headache or other signs of meningeal ir-

ritation, we may safely consider the case as one of suppurative labyrinthitis. On the other hand, if headache or signs of meningeal irritation are absent, it is not at all dangerous to wait, even though it be a case of suppurative labyrinthitis.

In considering the differentiation, a fact worth bearing in mind is that the suppurative labyrinthitis is much more frequent than the serous. Although serous labyrinthitis may be the precursor of the suppurative form, every attack of the suppurative labyrinthitis need not be preceded by an attack of serous labyrinthitis.

I, B.—CIRCUMSCRIBED LABYRINTHITIS.

Circumscribed labyrinthitis, secondary to middle ear suppuration, occurs with the bony labyrinth intact and too when not intact. When intact we have either a metastatic or toxic condition from extension of the infection or the toxins of infection by way of the lymph vessels or by a thrombotic condition of the mucous membrane of the middle ear extending through the vascular system of the bone into the labyrinth. (Macewen, *Infectioes Erkrankung* u. s. w., Wiesbaden, 1898.) More often circumscribed labyrinthitis occurs when the labyrinth capsule is not intact; that is to say, when there is some destructive process (caries, necrosis, osteoporosis or pressure atrophy, etc.) of the labyrinth capsule, due to cholesteatoma or tuberculosis; the latter condition especially in the first few years of life. The most frequent site is the prominence of the horizontal semicircular canal; the next most frequent site is the oval window; less frequent, the promontory; however, any part of the external wall may be involved. These latter processes lead to an opening in the capsule which permits the entrance and growth of granulations into the labyrinth and finally in some cases to destruction of the entire membranous labyrinth. These are the more frequent conditions, besides which we have empyema of the saccus endolymphaticus, described by Koerner and others.

We will not enter into a discussion of the pathology of these various lesions, concerning which the reader is referred to the works of Koerner, Hinsberg, Freytag, Heine, Neumann, Alexander, Ruttin and others. We shall discuss rather the symptomatology and diagnosis.

We may conveniently divide the circumscribed lesions of the labyrinth, according to their location, into those involving the cochlea and those involving the nonacoustic labyrinth according to the symptoms and reactibility of the part involved, into the irritative and destructive.

Irritative circumscribed lesions may, under favorable treatment, remain circumscribed or may (rarely) entirely disappear; however, when neglected the irritative processes usually becomes a circumscribed destructive or diffuse destructive process of the labyrinth.

From the above it is readily seen that we may, therefore, have:

I.—Circumscribed irritative lesion of the cochlea.

II.—Circumscribed destructive lesion of the cochlea.

III.—Circumscribed irritative lesion of the nonacoustic labyrinth (vestibule or semicircular canals).

IV.—Circumscribed destructive lesion of the nonacoustic labyrinth (vestibule or semicircular canals).

It should not be a difficult task to differentiate these four conditions from each other and from labyrinth suppuration.

I, B, I.—CIRCUMSCRIBED IRRITATIVE LESION OF THE COCHLEA may show most or all of the following characteristics:—

By Otoscopic examination:

(a) Polyps or granulations on the promontory, or exposed bone as determined by the use of the probe, with or without associated cholesteatoma.

Clinically:

(b) Tinnitis.

(c) Marked impairment of hearing for all tones if the entire cochlea is involved, or more especially the high tones if the lower cochlear whorl alone is involved.

(d) Bone conduction shortened (characteristic for all internal ear conditions).

(e) Very positive Gellé; that is to say, with compression of air in the external canal the bone conduction is shortened very much out of proportion to the normal (a condition opposite to that found in otosclerosis).

(f) Rarely scotoma, elicited by examination with Bezold's continuous chain of forks.

(g) Absence of symptoms from the vestibular apparatus with quite normal reactions to the caloric, turning and galvanic tests.

I, B, II.—CIRCUMSCRIBED DESTRUCTIVE LESIONS OF THE COCHLEA show by otoscopic examination:—

(a) Same as in condition I, in addition perhaps roughened bone, small sequestra or fistula in the region of the promontory.

Clinically:

(b) *Absolute deafness* for some or all tones, depending upon the extent of the lesion.

(c) Bone conduction very much shortened.

(d) Gellé, *i. e.*, compression and aspiration produces no effect upon the already very short bone conduction.

(e) No symptoms or signs from the vestibular apparatus.

I, B, III.—CIRCUMSCRIBED IRRITATIVE LESION OF THE NONACOUSTIC LABYRINTH. As previously stated the favorite location of this lesion is the horizontal semicircular canal or the region of the oval window. The otoscopic examination may reveal polyps or granulations, with or without involvement of the bone in the antrum or attic-antrum region. Clinically the symptoms are:

(a) Vestibular vertigo with its accompanying phenomena, nausea and vomiting, depending upon the intensity of the vertigo.

(b) Spontaneous nystagmus *toward the diseased side.* Horizontal if the horizontal semicircular canal is the seat of the irritation; rotatory, if the region of the oval window is the seat of the irritation. The nystagmus is pronounced during the attacks of vertigo but evident also between attacks.

(c) Increased reaction of the nonacoustic labyrinth, especially to turning and galvanism. The after-nystagmus to the *diseased* side may last from 35 to 100 seconds, while the after-nystagmus to the *well* side remains approximately normal or but slightly increased, 20 to 25 seconds.

The galvanic reaction shows nystagmus to the diseased side, when the kathode is to the diseased ear, with a much weaker current than is required to produce a nystagmus to the well side when the kathode is applied to the well ear. In making the galvanic test it is well to have the patient look straight ahead, since in this position the spontaneous nystagmus, which may be present when the patient looks to the sides, is perceptibly diminished. In exceptional cases only does the nystagmus persist when the patient looks straight ahead.

The caloric reaction test is purely a qualitative test and has no value in determining the degree of irritabiilty of the labyritnh in any case; however, it plays an important role in the diagnosis of the next (IV) class of cases.

(d) Compression and aspiration nystagmus; also known by some

writers as the fistula symptom (Barany). This symptom, when present, is a symptom of importance. If the lesion is in the horizontal semicircular canal, we have by compression, a horizontal nystagmus to the same side, and by aspiration horizontal nystagmus to the opposite side. If the lesion is in the region of the oval window or the vestibule, we have by compression rotatory nystagmus to the opposite side, and by aspiration rotatory nystagmus to the same side. We also obtain this latter (rotatory) form of compression and aspiration nystagmus in cases of dislocation of the stapes plate, so long as the labyrinth remains reactive.

(e) Acoustic symptoms, other than those belonging to the middle ear suppuration, are negative.

I, B, IV.—CIRCUMSCRIBED DESTRUCTIVE LESION OF THE NONACOUSTIC LABYRINTH presents the same general otoscopic picture as in condition III. besides the following clinical signs:

(a) Vestibular vertigo, with or without nausea and vomiting, according to the stage of the process. In the early stages the vertigo is considerably more intense than in condition III.

(b) Nystagmus, horizontal or rotatory, according to the location and extent of the process, *but always more marked to the well side* (similar to that met with in labyrinth suppuration and opposite to that met in irritative lesions of the static labyrinth).

(c) More or less positive disturbance of equilibrium, similar to that found in labyrinth suppuration (the early and the late forms).

(d) Caloric examinaiton is negative when the entire nonacoustic labyrinth is destroyed; partially negative* when a part only is destroyed. Relatively the same may be said for the turning and galvanic reactions.

The differentiation of the different forms of circumscribed labyrinthitis from each other and from labyrinth suppuration should not

*I say "partially negative" wittingly. As an illustration—it was my good fortune to be able to make repeated examinations of a case with an operative injury of the horizontal semicircular canal; immediately after the injury the patient exhibited all the signs and symptoms of acute destructive lesion of the static labyrinth with negative reactions, due no doubt to loss of endo- and perilymph. After ten days or two weeks the reactions began to reappear; though diminished, they were positive. Reactions from the horizontal semicircular canal remained negative while the reactions from the vertical canals remained positive by the caloric examination as well as by turning and galvanism.

be difficult. The differentiation of the irritative from the destructive form should be quite easy. There is but one of the four conditions which might be confounded with labyrinth suppuration and that is the circumscribed destructive lesions of the static labyrinth, and then only in those very old chronic cases where the hearing has been previously so greatly impaired that it may be mistaken for absolute deafness. In other words a case where through long continued suppuration, a gradual destructive process of the cochlea had taken place, when suddenly the patient develops an *acute* destructive lesion of the static labyrinth. Such cases are not so rare. The question is decided by the hearing tests; the slightest rests of hearing excludes diffuse labyrinth suppuration.

II. AFFECTIONS OF THE BONY LABYRINTH SECONDARY TO MIDDLE EAR SUPPURATION.

These are usually erosions, fistulæ, small areas of necrosis, with or without granulations and frequently associated with cholesteatoma or tubercular granulations. Any part of the labyrinth capsule may be involved, but more often the external wall. In cases where there is a necrotic piece of the capsule not yet loosened from the surrounding bone there may be an absence of symptoms until by manipulation with instruments or by pressure of water syringed into the middle ear, the necrotic piece is pressed inward against the membranous labyrinth when the patient, if the necrotic bone be in the region of the static labyrinth, experiences sudden vertigo with its accompanying phenomena; when in the region of the promontory, sudden tinnitus with or without deafness.* Similar symptoms may be experienced at any time during the attempt at extraction of polyps. The tearing away of a polyp with a snare may bring with it a small necrotic portion of the labyrinth capsule (such a case having been witnessed by the writer).

Affections of the bony labyrinth are sooner or later associated with some affection of the membranous labyrinth, either circumscribed or diffuse (see above).

The affections of the labyrinth capsule present three stages: first, —*Invasion Stage,* before symptoms are evident and the membranous

*Deafness with tinnitus would seem rather paradoxical, nevertheless their coexistence is not so rare in acquired deafness. (See Dr. Alice V. Mackenzie. "Galvan. Akustikus React," *Wiener Klin. Wochenschr.,* 1908, Nr. 11.)

labyrinth is intact and the reactions are normal. Second,—*Manifest Stage;* symptoms of irritation of the membranous labyrinth are evident. Third,—*Final Stage;* may be limitation of the process with resolution or may be the spreading of infection to the entire membranous labyrinth (labyrinth suppuration) or beyond the labyrinth to the meninges and brain.

Of the different affections of the labyrinth capsule, fistula of the horizontal semicircular canal at the prominence is the most frequent and easiest recognized, and for this reason we shall give it some special attention (see Neumann, "Uber einen Fall zircumscripter Labyrintherkrankung," Oct., 1907, *Monatschr. f. Ohrenheilk*).

Fistula of the prominence of the horizontal semicircular canal is generally but not invariably due to erosion from cholesteatoma. The fistula in its earliest stage may, as previously stated, be free of symptoms because of a quite normal membranous labyrinth. In the later stages the membranous labyrinth may be considererably involved, even to the extent of destruction, evident by negative reactions.

A characteristic sign of fistula, so long as the membranous labyrinth is intact, is the so-called fistula symptom of Barany, better known by other writers as the compression and aspiration nystagmus. Alexander and Lasalle, "Ueber den durch Luft Druckveränderungen aus lœsbaren Nystagmus und das Fistel Symptom," *Wiener Klin. Rundsch.,* 1908). This symptom is negative in late cases after the membranous labyrinth is destroyed. The production of this nystagmus is really nothing more than the clinical application of Ewald's experiment made upon the semicircular canals of animals. (See Barany on "Physiologie und Pathologie des Bogeng. Apparat.") The examination is made with a Gellé balloon or ordinary Politzer bag fitted with rubber tubing, on the end of which is attached a perforated spherical or olive pointed hard rubber tip. The tip is fitted into the external canal firmly enough to prevent the escape of air. Compression of the balloon causes increased air pressure in the canal and middle ear cavity. A positive reaction is denoted by a horizontal nystagmus to the same side by compression and to the opposite side by aspiration.

This symptom is positive in the first and second stages and negative in the final stage. Aside from this symptom the first stage of fistula presents negative symptoms and signs.

The second stage will show this sign plus the signs of circumscribed

irritative lesion of the labyrinth (see above). In the third stage, the compression and aspiration nystagmus is negative and the other signs and symptoms are those of circumscribed destructive lesion of the labyrinth, or if there has been a spreading of infection then we have the symptoms and signs of diffuse destruction (labyrinth suppuration).

III. AFFECTION OF THE PERILABYRINTHINE SPACES.

*PERILABYRINTHITIS is a process which involves the perilabyrinthine tissue and is favored by a pneumatic condition of the cells in the pyramid surrounding the labyrinth capsule. These cells are in direct communication with and form a part of the mastoid cells. Severe constitutional disturbances and especially diabetes favor the extension of mastoiditis into these perilabyrinthine mastoid cells with resulting partial or complete sequestration of the bony labyrinth. Perilabyrinthitis may be associated with acute or chronic suppuration of the middle ear. The secondary dangers, meningitis and brain abscess, are not so great in this as in labyrinth suppuration.

The symptoms and signs of perilabyrinthitis are very like those of labyrinth suppuration; however, there are points of difference which make it possible for us to make a differential diagnosis between these two processes and are as follows:

LABYRINTH SUPPURATION.	PERILABYRINTHITIS.
1.—More often secondary to *chronic* middle ear suppuration.	1.—More often secondary to *acute* middle ear suppuration.
2.—The process begins *suddenly.*	2.—The process begins more *gradually.*
3.—May be, but not necessarily, associated with mastoiditis.	3.—*Always* associated with mastoiditis.
4.—Otoscopic findings *not* characteristic.	4.—Otoscopic findings in advanced cases always show *necrotic bone.*
5.—Associate facial palsy *the exception,* and when present has no direct connection with labyrinth suppuration.	5.—Facial palsy *the rule,* since the facial canal is surrounded by the same perilabyrinthine cells as is the labyrinth itself.

*I prefer the term Perilabyrinthitis to that of Paralabyrinthitis used by most of the German authors.

6.—*As a rule,* associated meningeal irritation or meningitis or other intracranial complications.	6.—Intracranial complications *the exception.*
7.—No direct relationship to systemic or constitutional diseases.	7.—Frequent association of systemic or constitutional diseases.
8.—The Weber test shows lateralization to the *well ear.*	8.—According to Neumann, Weber lateralized to the *diseased ear.* However, with writer, this is still an open question.

DIFFERENTIATION OF LABYRINTH SUPPURATION FROM CEREBELLAR ABSCESS.

THE preceding paper upon the subject of Differential Diagnosis dealt only with the surgical diseases of the labyrinth secondary to middle ear suppuration, the writer having reserved this, a separate paper, for the differentiation of labyrinth suppuration from cerebellar abscess. Before taking up the differentiation it will be well first to go briefly over the subject of cerebellar abscess, just as we have in our first paper gone briefly over the subject of labyrinth suppuration.

To begin with, let us recall the facts, first, that the physiology of the cerebellum is still a somewhat unsettled problem; secondly, that the symptomatology of cerebellar diseases (including abscess) is very variable. In looking over the literature upon the subject of cerebellar abscess, one is impressed with the disagreement of authors, no two of them agreeing upon any set of pathognomonic or even cardinal signs. The nearest approach to such signs is to be found under the heading, "Zerebellar Nystagmus," in Neumann's work upon this subject, "Der Otitische Kleinhirn Abscess," 1907. The above facts are a sufficient reason for our inability in some cases to make exact diagnoses.

The cause of cerebellar abscess is suppuration of the middle ear, according to Oppenheim in almost one-half the cases, and according to Ghon (in a paper read before the American Medical Association of Vienna, in 1907) in about two-thirds of the cases.

Otitic cerebellar abscess occurs somewhat less than half as frequently as otitic temporal-lobe abscess, or as 198 is to 428 (Heimann, *A. f. O.*, bd. 66 and 67).

Cerebellar abscesses vary in size from that of a hazelnut to that of a hen's egg and larger. They are generally single, but may be multiple; however, Neumann believes that the apparently multiple abscesses are nothing more than so many extensions of a single abscess. There is no direct proportion between the size of the abscess and the symptoms it may produce.

Cerebellar abscess may run an acute or chronic course. The course is dependent upon the virulence of the infecting micro-organism and

the presence or absence of a pyogenic membrane. The diplococcus of Fraenkel-Weichselbaum is prone to form a capsule, while the anærobic bacteria do not. The latter tend also to the formation of very fetid smelling abscesses (Ghon, *Zentralblatt f. Bakt.*, 41, 1906).

Otitic cerebellar abscess may result from acute or chronic middle ear suppuration. From the combined statistics of Okada, Heimann and

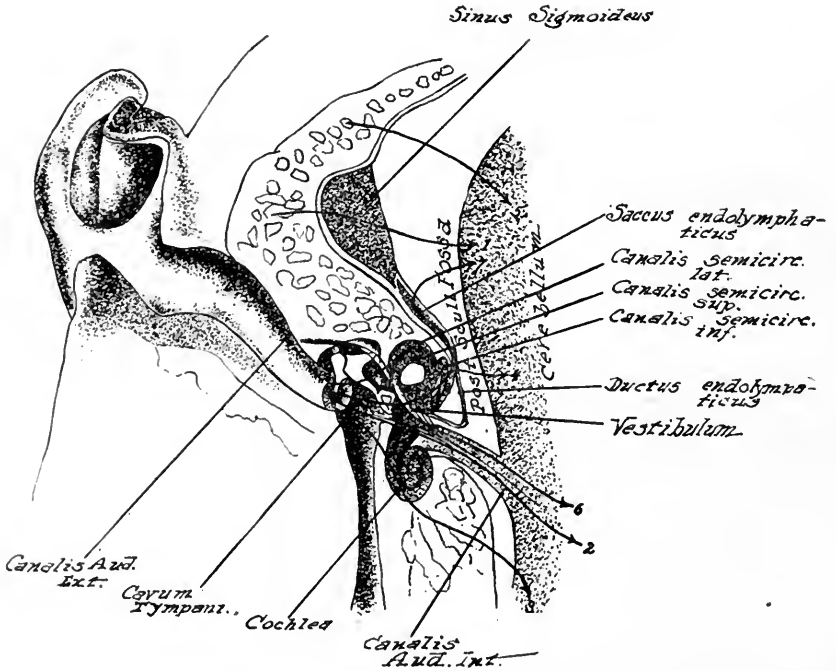


Figure 1. Half schematic horizontal cut through the right ear, showing the routes of infection from the middle ear to the posterior skull fossa. (By Alexander). Almost normal size.

Neumann in 869 cases, 82 per cent. resulted from chronic and 18 per cent. from acute middle ear suppuration.

The routes of infection, according to Politzer, in the order of their frequency are: 1, labyrinth (in cases following labyrinth suppuration); 2, posterior surface of the pyramid (in cases of caries and necrosis); 3, lateral sinus wall (in case of sinus phlebitis); 4, median wall of the mastoid. According to Alexander, the route of infection to the posterior fossa is thru *preformed and nonpreformed ways.* To better show these various routes the reader is referred to figure 1, half schematic,

by Alexander in Professor Hochenegg's "Lehrbuch der Chir. und Oper.," 1906, and more recently "Die ohrenkrankheiten im Kindesalter 1912 by Alexander.

The preformed ways are thru the labyrinth along the nerve packet of the canalis auditorius interna, indicated in Fig. 1 by the heavy line 2; along the facial canal, indicated by heavy line 6; along the ductus and saccus endolymphaticus, indicated by heavy line 7.

The nonpreformed ways are: sinus sigmoideus, indicated by line 1; thru the cochlea, indicated by line 3, or thru the semicircular canals, indicated by line 4; thru the median wall of the mastoid behind the sinus, indicated by line 5. Infection of the posterior fossa may also take place thru a combination of two or more of these ways.

Infections along preformed ways, with the exception of the ductus and saccus endolymphaticus (Alexander, Jansen, Neumann), are more prone to produce meningitis (diffuse or circumscribed) than infection thru nonpreformed ways. On the other hand, infections thru the ductus and saccus endolymphaticus and the nonpreformed ways are more prone to produce extradural and cerebellar abscesses. It must not be forgotten that there are exceptions to these rules.

Otitic cerebellar abscess is more liable than not to be associated with other complications, which accounts for the variability and confusion of the symptoms already mentioned. These complications are:

1. Sinus phlebitis. Found especially in cases of cerebellar abscess following *acute* middle ear suppuration; 8 out of 19 cases, or 40 per cent., according to Neumann.

2. Extradural abscess. Politzer and others have found this complication especially frequent in cases of cerebellar abscess following *chronic* middle ear suppuration. The dura is thickened and adherent, and frequently there is a communicating fistula between the extradural and cerebellar abscesses. Koerner fouud this fistulous communication in 42 per cent. of otitic brain abscesses irrespective of location.

3. Subdural abscess (Heine) found in *acute* cases; however, the percentage is small.

4. Labyrinth suppuration is found in more than one-half of the cases of cerebellar abscesses following *chronic* middle ear suppuration (combined estimate of several authors), but is *never* found in acute cases (Neumann). Labyrinth suppuration resulting from *acute* middle ear suppuration is more apt to be followed by meningitis.

5. Circumscribed pachyleptomeningitis and serous meningitis (Alexander, Neumann, Koerner).

I have not included, in the above grouping, diffuse suppurative meningitis, since this is rather the result than an associated complication of cerebellar abscess.

Heimann (*A. f. O.*, 66, 251) in a conservative statement claims that almost 50 per cent. of otitic cerebellar abscesses are uncomplicated. This may be taken as a negative statement, acknowledging that somewhat more than 50 per cent. are complicated. Most authors accept even larger figures.

Cerebellar abscesses present four stages: 1, initial stage; 2, latent stage; 3, manifest stage; 4, terminal stage.

1. Initial stage. This is the stage of primary infection, which takes place rather abruptly and is accompanied by a change in char-

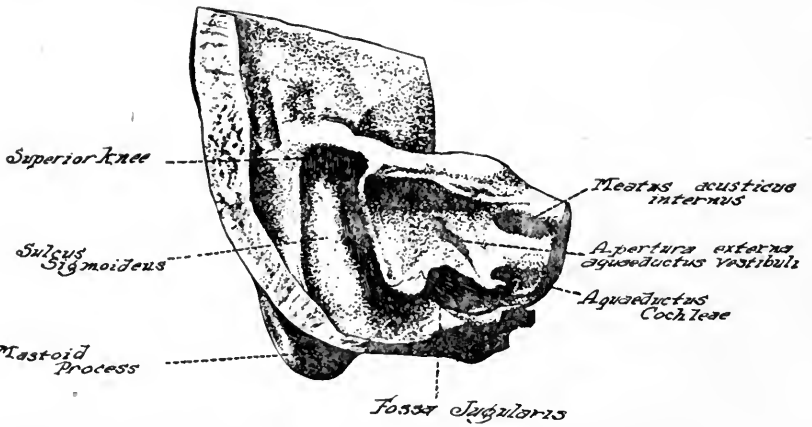


Figure 2. Posterior surface of the temporal bone. (By Alexander). Almost normal size.

acter or amount of secretion from the middle ear (Alexander); initial chill with, as a rule, not very high fever; vertigo, rather inconstant; headache, especially occipital but may be frontal; retroauricular tenderness and general malaise. These symptoms are more or less pronounced, altho frequently forgotten by the patient, who may present himself for the first time during the manifest stage. The symptoms of the initial stage are frequently attributed to some obscure intercurrent, so called bilious, attack. The initial stage is brief and subsides after a few days, when the condition goes over into a more or less prolonged latent stage.

2. Latent stage. This is the intervening stage before the third or manifest stage. During the latent stage there is more or less dull

headache, localized retroauricular tenderness; in addition, according to Politzer, moderate amount of prostration, apathy, pallor of the face, loss of body weight, loss of appetite; according to Oppenheim, moderate elevation of temperature, occasional epileptiform or hysterical fits, psychic disturbances, apathy and melancholia. These symptoms, according to the opinion of the writer, are not especially indicative of cerebellar abscess, however they may suggest it.

To the symptoms just enumerated there may be in addition symptoms of labyrinth suppuration or extradural abscess when either of these complications are found associated with *chronic* middle ear suppuration, or the symptoms of sinus phlebitis when this complication is found associated with *acute* middle ear suppuration.

Referring to complicating extradural abscess, the headache and circumscribed tenderness may be identical with that found in cerebellar abscess. According to Alexander, the location of this tenderness is 4 or 5 cm. behind the auricle, and the spot is so circumscribed that it may be covered with the ball of the thumb. Alexander (Lehrbuch der Chir. und Oper., Prof. Hochenegg) advises prompt operation for *all* cases of extradural abscess, upon the ground that if not operated there is great danger of meningitis, sinus phlebitis or brain abscess resulting.

Since it is recognized that a fair percentage of cerebellar abscesses follow extradural abscess and labyrinth suppuration, it is logical treatment to expose the dura and favor drainage in case of extradural abscess and to remove a sufficient part of the labyrinth for the same purpose in case of labyrinth suppuration before the cerebellar abscess or other intracranial complications have had time to develop. The efficiency of this form of radical treatment has been already demonstrated by the steady falling off in the number of cases of cerebellar abscess in the Politzer klinik during the last few years, where this line of treatment has been carried out. Alexander claims the same results for the Polyklinik in Vienna.

The latent stage may last from a few weeks to several months, and even longer (Oppenheim, MacEwen).

3. Manifest stage. This stage represents that of the fully developed abscess. In this stage the symptoms are pronounced. The headache (occipital or frontal), occipital tenderness, vertigo, vomiting, lassitude, apathy, characteristic pallor of the face, inform us that the patient is suffering some severe intracranial complication, possibly

cerebellar abscess. This condition lasts, as a rule, a few weeks or longer, in exceptional cases but a few days only; during this time the patient diminishes rapidly in body weight, even out of proportion to the diminution of nourishment taken. This stage passes more or less abruptly over into the *terminal* stage.

4. Terminal stage. This stage is generally brief and almost invariably results fatally.

In exceptional cases spontaneous recovery has been observed thru rupture of the abscess externally into the middle ear cavity (Bruns, Huguenin, Wilde, Pollak and others); also thru resorption of the contents of abscess (Braun, Brieger, Rheinhard); however, Politzer believes that most of these cases were rather cases of extradural abscess.

The terminal stage of cerebellar abscess is due more frequently to rupture of the abscess into the leptomeningeal space or the ventricles with resulting diffuse suppurative meningitis in the first instance or acute hydrocephalus from closure of the foramen Magendi with the products of the abscess in the second instance, less frequently to sudden increase of volume of the abscess causing pressure upon the medulla oblongata, and thereby death from sudden paralysis of respiration, while the heart tends to continued action for hours, especially where the respiration has been artificially continued. (Barker, *British Medical Journal*, 1902; Fliess, *Deutsch. Med. Wochenschr.*, 1903.) Knapp reports a case of continued heart action for forty-eight hours after respiration had been artificially continued.

Other, but still less frequent, causes of death are toxic coma, encephalitis serosa in the area surrounding the abscess, sinus phlebitis (pyemia) and pneumonia.

SYMPTOMATOLOGY.

Takabataka (*Z. f. O.*, 46, 236) has demonstrated that there is a lack of relationship between the size of the abscess and the symptoms it may produce.

The symptoms of cerebellar abscess are those belonging (1) to the primary middle ear supuration; (2) to the cerebellum itself (focal symptoms); (3) to neighboring or distant parts (so called "durch fernwirkunk" symptoms of the German authors), the medulla, pons, cerebral hemispheres, intracranial nerve roots of the posterior fossa (Koerner) and the upper part of the cervical cord (MacEwen); (4) to increased intracranial pressure; (5) to associated complications, especially labyrinth suppuration, extradural abscess, encephalitis, sinus

phlebitis and meningitis; and (6) to toxic origin (Neumann) due to the absorption of the products of the abscess. It is often difficult to isolate a symptom and tell positively to which of these it is due. Accordingly I shall not make any attempt at classification of the symptoms as Koerner, Neumann and a few others have done, but shall content myself rather with the enumeration of the symptoms as found in a case of cerebellar abscess. Many of these symptoms are accepted by most authors and denied by others. I shall classify the symptoms under three headings:

I. The more frequent symptoms.
II. The less frequent symptoms.
III. The special symptoms.

Wherever it is possible, I shall refer to their relative value and possible origin.

I. THE MORE FREQUENT SYMPTOMS.

These together with the special symptoms are the more important symptoms found in cerebellar abscess. They are:

1. **Headache.** Occipital headache upon the affected side is the most characteristic and typical headache of cerebellar abscess. Less frequently there is frontal headache. Krause explains the frontal headache as reflex from the ramus recurrens Arnoldi, branch of the fifth nerve (which supplies the tentorium) to the ramus meningeus anterior which supplies the dura of the anterior fossa. Very rarely or never is there parietal headache present as in case of temporal lobe abscess.

2. **Pressure or percussion tenderness** in the occipital region of the affected side. This tenderness is more often circumscribed, but may be diffuse. This symptom as well as the preceding (occipital headache) is no more characteristic for cerebellar than for extradural abscess, as pointed out elsewhere.

3. **Rigidity of the neck,** more especially the nape, is frequently combined with inclination of the head to the diseased side. This symptom is held by Alexander and Von Beck as a very important one. Neumann, on the contrary, believes this symptom is not characteristic for uncomplicated cerebellar abscess. A characteristic feature of this symptom is that the patient supports the head with the hands during attempts at movements. The frequency of this symptom in cerebellar abscess has been observed by the writer.

4. **Homolateral hemiparesis and hemianesthesia** in contradistinction

to the contralateral found in temporal lobe abscess. In a case of Uffenorde's (*A. f. O.* 67, 179 and 189) convulsions in the arm of the same side. The cause of the homolateral hemiparesis is ascribed by some authors to pressure upon the opposite pyramid at the foramen magnum, while others (Pineles, Monokow) believe it is purely a focal symptom from the cerebellum. The writer inclines to the latter explanation as the more possible and logical one.

5. **Cerebellar ataxia,** observed by innumerable authors, seems to be purely a focal symptom (Nothnagel ascribed its origin to pathologic causes located in the worm of the cerebellum), especially noticeable in the extremities of the affected side. The trunk muscles are also frequently involved. In this connection Babinski has noted a peculiar phenomenon, which he terms "asynergie cerebelleuse." This phenomenon manifests itself when the patient attempts to walk: the legs proceed forward in the usual manner but the trunk does not follow; remaining behind, giving the patient a peculiar attitude. This phenomenon manifests itself also when the patient attempts to walk backward, the relative position of the legs and trunk assuming the reverse position to that found when walking forward. Babinski believes the symptom to be a very important one in cerebellar abscess.

6. **Optic neuritis,** or choked disc, is found more frequently in cerebellar abscess (Politzer, Hinsberg, Koerner, Neumann and others) than in temporal lobe abscess. Zaufall claims that he never failed to find some fundus changes in cerebral complications of *chronic* middle ear suppuration; the changes however were often very slight. These changes include unsharpness of the disc, overfilling of the veins and increased tortuosity of the vessels. On the other hand, Jansen (*A. f. O.,* 36), Koerner (*Z. f. O.,* 23) and Bezold (*Handbuch ges. Augenheilk.,* von Graefe Saemisch, 1903) have claimed similar changes for simple empyema of the mastoid cells, which cleared up promptly after simple mastoid operation. Notwithstanding, the concensus of opinion is that neuritis or choked disc is a very frequent sign of cerebellar abscess

7. **Impairment of intellect** (slow cerebration, MacEwen) together with apathy, somnolence, stupor and finally coma, form a symptom group quite characteristic for cerebellar abscess. Alexander lays great stress upon the somnolence. The slow cerebration may be manifested even during the latent stage, while somnolence belongs rather to the later stage. Neumann, while recognizing these as important symptoms found in cerebellar abscess, places them among those produced by the effects of the abscess upon more distant parts of the brain.

8. **Slowness of the pulse rate,** held by MacEwen and Barr as one of the most important signs of cerebellar abscess, the pulse rate diminishing to thiry beats per minute and less. Arythmia of the pulse (Neumann) is quite characteristic; notwithstanding, we find the same character of the pulse in many other conditions (encephalitis, tumor cerebri, etc.) where the intracranial pressure is greatly increased.

9. **Trophic disturbances,** as evidenced by rapid loss of body weight out of proportion even to the diminution of nourishment taken, as a result of anorexia. This symptom is so characteristic and frequent that Okada was able to detect it in 37 of his 40 cases.

10. **Anarthria and dysarthria** (Fimmer, dissertation, Leipsic, 1905), supposed to be the result of pressure of the abscess or the effects of the toxins upon the medulla oblongata. These symptoms, no matter how produced, have been repeatedly observed in association with cerebellar abscess, and the writer considers them, when present, as symptoms of relative importance in cerebellar abscess.

II. THE LESS FREQUENT SYMPTOMS.

Let us next consider symptoms belonging to group II. These symptoms, tho less frequent than the former, can hardly be considered less important since they have been observed and reported by equally trustworthy observers. They are:

11. **Deviation conjugee.** Neumann (der Otit. Kleinhirn Abscess, p. 28) observed this symptom in four of his cases. Relative to this sign the writer wishes to note that it is not necessarily a cortical one, but instead is a reflex in a *comatose* patient produced by any irritation—in Deiter's nucleus or in the posterior longitudinal bundle—which would produce nystagmus in a conscious patient. This deviation represents but one phase of the nystagmus, observed by Barany, the writer and others in patients under general anesthesia, who in a wakeful condition show nystagmus.

To illustrate: If a patient has an irritative lesion in the semicircular canals, vestibular nerve or Deiter's nucleus of the right side, he will exhibit a rotatory nystagmus to the right side. The nystagmus is furthermore rhythmic and, as has been pointed out, is composed of a rapid movement of the eyeballs to the right and a slow movement to the left. The slow movement represents the vestibular reflex, while the rapid movement represents the voluntary cortical act. Should the same patient be put under a general anesthetic, the voluntary cortical movement is suppressed and there remains but the reflex movement

alone, the eyes assuming the extreme lateral position to the opposite (left) side; in other words, we have deviation conjugeé to the side opposite the irritation, as found in cases of cerebellar abscess.

I agree with Neumann that the sign, when present, is an important positive one, and furthermore I believe the sign may be observed more frequently than it really is if carefully looked for in all cases.

12. **Herpes labialis,** observed by Schmiegelow (*Archives Internat. de Laryng.,* XIX, 337) in four cases out of nineteen of brain abscess, irrespective of location. Observed also by Koerner (*Z. f. O.,* 44) and freely quoted by others.

13. **Homolateral hypoglossus paresis** observed by Uffenorde (*A. f. O.,* 67, 179).

14. **Glycosuria,** observed by Frey (*Z. f. O.,* 58, 171), also by Grunert and MacEwen.

15. **Retention and incontinence of urine and albuminuria** have been observed by Brieger, Oppenheim and MacEwen.

The symptoms thus far considered belong more or less distinctively to cerebellar abscess and not to labyrinth suppuration. The presence or absence of these symptoms are important ones in the differential diagnosis.

We shall next consider a group of symptoms (III. Special symptoms) common to both conditions, symptoms which are responsible for the resemblance of the two processes, and which make necessary the differential diagnosis of labyrinth suppuration from cerebellar abscess. In considering these symptoms, we shall at the same time consider them from the standpoint of the differential diagnosis.

III. SPECIAL SYMPTOMS.

These are **vertigo, nystagmus** and **equilibrium disturbance.** They form a symptom complex which because of their association and dependence upon one another, both in labyrinth suppuration and cerebellar abscess, must be considered together. These symptoms may be considered as the several manifestations of the one phenomenon which, when most pronounced, manifests itself still further with **nausea** and **vomiting.**

Alexander has noted that every attack of vertigo, independent of cause, is accompanied with rhythmic nystagmus, and I would add, too, with equilibrium disturbances.

On the other hand, rythmic nystagmus may be observed without vertigo, as illustrated by the low degrees of galvanic nystagmus ob-

tainable without the subjective sensation of vertigo (a fact first pointed out by the writer).

Likewise, equilibrium disturbance may be obtained in diseases of the centripetal tracts to the cerebellum without vertigo, providing the process is not a very acute one. Panse (Schwindel, 1902), Mackenzie (*A. f. O.*, 1908).

The opinion of Marburg is that the symptoms of vertigo, nystagmus and equilibrium disturbance in case of cerebellar abscess arise from irritation of Deiter's nucleus and necessarily are of the same character (so called vestibular) as those found in irritative lesions of the labyrinth or vestibular nerve. On the other hand, Alexander (*M. f. O.*, 1906) claims that the character of the nystagmus in cerebellar abscess is distinctly horizontal and of *long* excursions in contradistinction to the vestibular nystagmus which is rotatory and of relatively *short* excursions.

Of more importance than the character is the *direction* of the nystagmus. Oppenheim, Koerner, Panse, and more recently Alexander, Neumann, Barany and others of the Politzer school have shown that tho direction of the nystagmus in cerebellar abscess may be to either side, in the vast majority of cases it is toward the diseased side.

In spite of the fact that labyrinth suppuration and cerebellar abscess present a marked resemblance so far as the vertigo, nystagmus and equilibrium disturbances are concerned, Neumann contends, and is amply supported by others, that these very symptoms, and more especially the nystagmus, are the symptoms which aid us most in the differential diagnosis.

It would seem that the difficulties of diagnosis would be much increased when we have a combination of the two conditions, but even here a differentiation in most cases is not so difficult as appears at first thought. In a case of combined labyrinth suppuration with cerebellar abscess the symptoms and signs of the more recent cerebellar abscess outweigh those of the longer standing labyrinth suppuration. If the symptoms and signs of cerebellar abscess are sufficiently pronounced to make a positive diagnosis, it remains only to determine the negative reactibility of the labyrinth to complete the diagnosis. In such a case (cerebellar abscess following labyrinth suppuration) there is always an appreciable lapse of time between the manifestations of these two processes. The patient, if conscious, will be able to give a history describing his attack of labyrinth suppuration (sudden deafness, vertigo with nausea, vomiting and equilibrium disturbance so pronounced that he

was compelled to seek the recumbent position, lasting over a period of several days) so characteristic that a mistake in diagnosis of labyrinth suppuration seems almost impossible. If in combination with the above history we are able to determine that deafness is absolute and the static labyrinth is nonreactive to one or more of the usual tests the diagnosis is certain. On the other hand, if we find the patient in a comatose condition, some member of the family or friend will be able to supply a history which, together with negative reactibility of the static labyrinth determined at the time of our examination, are quite sufficient for us to make a positive diagnosis. I might add that these latter are the cases which are prone to show the deviation conjugeé (see above) to the opposite (well) side.

The nystagmus of labyrinth suppuration and that of cerebellar abscess differ not only in character and direction, as already pointed out, but, too, in intensity. This intensity differs with the stage of the processes. In labyrinth suppuration it is most intense at the onset and then *diminishes* constantly with an almost mathematic precision; on the contrary, in cerebellar abscess the intensity tends to *increase*, is rather inconstant, and with frequent intermissions. As a result we find that the nystagmus of cerebellar abscess is rarely so intense as that of *early* labyrinth suppuration (first few days). On the other hand, it is always more intense than that of *late* labyrinth suppuration (several weeks or months after the acute attack). Since cerebellar abscess does not develop until long after the nystagmus of early labyrinth suppuration has had time to subside, it must follow that a marked nystagmus at this time must be due rather to cerebellar abscess,—and not to labyrinth suppuration.

Let us consider next the nystagmus from another standpoint. A case with the history and findings of middle ear suppuration presents itself with nystagmus to the opposite (well) side. There are three possibilities, namely: (1) labyrinth suppuration, (2) cerebellar abscess, (3) combined cerebellar abscess with late labyrinth suppuration.

(1) Labyrinth suppuration (see first paper) would be determined by the absolute deafness and the negative reactibility of the nonacoustic labyrinth, together with the absence of symptoms of cerebellar abscess.

(2) Cerebellar abscess (exceptionally rare) would be decided by the positive hearing and the positive reactibility of the static labyrinth, together with positive symptoms of cerebellar abscess.

(3) Combined cerebellar abscess with labyrinth

suppuration (very rare) would be decided by the negative hearing and the negative reactibility of the static labyrinth with positive symptoms of cerebellar abscess.

A second case, with the history and findings of middle ear suppuration, may present itself with nystagmus to the same (diseased) side. We have again three possibilities, namely: (1) irritative lesion of the labyrinth, (2) cerebellar abscess, (3) combined cerebellar abscess with late labyrinth suppuration.

(1) Irritative lesion of the labyrinth (see former paper) would be decided by the positive hearing, the positive caloric reactibility and the *increased* reactibility of the labyrinth to turning and the galvanic current, with perhaps (dependent upon the presence of fistula) compression and aspiration nystagmus.

(2) Cerebellar abscess would be decided by the positive hearing and the more or less positive reactions from the static labyrinth, and in addition the presence of symptoms of cerebellar abscess referred to in the preceding pages.

(3) Combined cerebellar abscess with labyrinth suppuration would be decided by the negative hearing and the negative reactibility of the static labyrinth, findings which alone are sufficiently characteristic for a positive diagnosis of a combination of these two processes. These findings, *i. e.,* nystagmus to the diseased side with negative reactions from the static labyrinth may be accepted as pathognomonic signs of combined cerebellar abscess and labyrinth suppuration.

The Vertigo of labyrinth suppuration like the nystagmus, is most pronounced at the onset of the attack and diminishes constantly with the same mathematic precision, however more rapidly than the nystagmus, and practically disappears in about a week, after which it reappears only upon making rapid movements of the head and then lasts but a few seconds.

The Vertigo of cerebellar abscess is rather less intense than that of early labyrinth suppuration (first week) but more intense than that of late labyrinth suppuration. In cerebellar abscess, instead of diminishing, the vertigo tends rather to increase with the progress of the abscess. Again, the vertigo of cerebellar abscess is more variable than that of labyrinth suppuration.

Nausea and vomiting invariably accompany the vertigo of early labyrinth suppuration, the nausea being the *more* pronounced of the two symptoms. In this instance the nausea is the result of the vertigo

On the other hand, in cerebellar abscess—altho the nausea and vomiting may at times result from the vertigo—it more often does not. In the latter instance (cerebellar abscess) nausea is *less* pronounced than the vomiting. The vomiting is more like the so called cerebral vomiting, forcible expulsive vomiting without nausea. Koerner attributes this cerebral vomiting to any condition which may produce increase of the intracranial pressure.

Equilibrium disturbance, like the nystagmus and vertigo of labyrinth suppuration, is most pronounced in the early stage, and is of the type already described (see former papers). The patient tends to fall laterally in the plane (frontal) of his nystagmus toward the *diseased* side. This falling in early labyrinth suppuration is an accompanying phenomena of the nystagmus; in other words, "reaction falling." (Barany, Phys. u. Path. des Bogeng. Appar., 1908.) On the contrary, the equilibrium disturbances of cerebellar abscess appear in the later stages. Altho the nystagmus in the two processes is to opposite sides, the tendency to fall in both cases is to the same side. In cerebellar abscess, since the nystagmus is to the diseased side, we *cannot* explain the falling to the diseased side as reaction falling the result of the nystagmus. It is probably due more to the hemiparesis and hemiataxia of the diseased side, as suggested by Alexander. This explanation receives further support in view of the fact that the so called "asynergie cerebelleuse" is really a form of ataxia of cerebellar origin.

In closing the subject of differential diagnosis of labyrinth suppuration from cerebellar abscess we shall, for the sake of convenience, summarize the more important differential symptoms in the form of a table (see below). Furthermore, since a differentiation of these two processes would not be complete without consideration of a third process, namely, combined labyrinth suppuration and cerebellar abscess, we shall include it in our table; besides, the frequency and importance of this third condition demand it. The table follows:

LABYRINTH SUPPURATION.	CEREBELLAR ABSCESS.	COMBINED LABYRINTH SUPPURATION AND CEREBELLAR ABSCESS.
1.—Absolute *deafness* of sudden onset.	1.—*Positive hearing* or rests of hearing.	1.—Absolute *deafness.*
2.—Vertigo intense at first, but constantly *diminishing*, with the sensation of objects rolling about the axis of vision.	2.—Vertigo less intense, but *increasing* with the progress of the abscess.	2.—History of having had vertigo of the type of early labyrinth suppuration which has diminished, again reappearing with increasing intensity.

LABYRINTH SUPPURATION.	CEREBELLAR ABSCESS.	COMBINED LABYRINTH SUPPURATION AND CEREBELLAR ABSCESS
3.—Pronounced spontaneous rotatory nystagmus to the *well* side in the early stages, but progressively *diminishing* in intensity.	3.—Spontaneous horizontal nystagmus of longer excursions than that of labyrinth suppuration (Alexander) to the *diseased* side or rotatory to either side, however in the vast majority of cases to the diseased side, *increasing* in intensity.	3.—Spontaneous rotatory nystagmus of labyrinth suppuration has almost disappeared and the nystagmus of cerebellar abscess type has appeared (see column to the left).
4.—Equilibrium disturbances *marked* in the *early stages* with tendency to fall to the *diseased* side (reaction falling). Less marked but apparent in the late stage.	4.—Equilibrium disturbances less marked than in early and more marked than in late labyrinth suppuration. Patient tends to fall to the *diseased* side because of muscular weakness and ataxia in the extremities of the diseased side (Alexander).	4.—Equilibrium disturbances of the same type as that found in cerebellar abscess (see opposite column to the left).
5.—Reactibility of the static labyrinth *negative.*	5.—Reactibility of the static labyrinth *positive.*	5.—Reactibility of the static labyrinth *negative.*
6.—Headache, when present, is *diffuse* over the lateral half of the affected side.	6.—*Circumscribed occipital* headache upon the affected side, or less frequent frontal headache.	6.—Same as that found in the opposite column to the left.
7.—No symptoms of cerebellar abscess.	7.—*Positive* symptoms of cerebellar abscess; localized occipital tenderness, rigidity of the neck, homolateral hemi-paresis and hemi-anesthesia, cerebellar ataxia, optic neuritis or choked disc, impairment of intellect (slow cerebration), great loss of body weight, bradycardia.	7.—*Positive* symptoms of cerebellar abscess outweigh those of the late labyrinth suppuration.
8.—Removal of the labyrinth is followed by improvement of the headache or other symptoms which may suggest meningitis.	8.—Removal of the labyrinth is not indicated; hence in this case no comparison can be made.	8.—Removal of the labyrinth *is not* followed by improvement as in the case of simple labyrinth suppuration. On the contrary, the symptoms of the cerebellar abscess are often increased by the trauma of the operation.

I have not discussed the value of the lumbar puncture in the differential diagnosis of these two conditions for evident reasons: First, because the contrast is not sufficiently great to be of any aid to us in the differentiation; both processes showing relatively the same conditions, namely, escape of cerebrospinal fluid with increased pressure, fluid slightly clouded containing few leucocytes and many lymphocytes, but no bacteria (except in case where the abscess has ruptured) and the fluid shows slight tendency to coagulation into delicate threads after long standing. Secondly, because the danger of rupture of the abscess from the sudden release of intracranial pressure is too great to warrant the procedure in case of cerebellar diseases.

The lumbar puncture is better adapted to, and is of more value in the differentiation of other processes.

THE DIFFERENTIATION OF LABYRINTH SUPPURATION FROM AFFECTIONS OF THE EIGHTH NERVE.

FROM the etiologic standpoint the most important diseased conditions demanding a differentiation from labyrinth suppuration are the diseases of the labyrinth secondary to middle ear suppuration; from the anatomic standpoint, or in other words from the standpoint of localization, are the diseases of the cerebellum (especially cerebellar abscess) and the diseases of the eighth nerve. The former (diseases of the labyrinth) were discussed and differentiated in the first paper upon Differential Diagnosis; cerebellar abscess in the following paper, and it remains for us to do the same with the diseases of the nerve in the present paper.

As is well known, a differential diagnosis of two or more diseased conditions implies first similarity, secondly dissimilarity. The similarity makes the differention necessary, and the dissimilarity makes it possible.

The *similarity* of symptoms and signs of destructive lesions of the labyrinth (deafness, vertigo, equilibrium disturbances, spontaneous nystagmus to the sound side and negative reactibility of the static labyrinth) to those of destructive lesions of the nerve are quite apparent; on the other hand the *dissimilarity* of the history and otoscopic findings are just as apparent. In spite of these apparent dissimilarities, we find cases where there is a possibility of confusion; a striking illustration of which was observed by the writer in a case of multiple neuritis involving the eighth nerve.

In this case a herpes zoster bleb on the tympanic membrane obscured the anatomic relationship and at the same time produced swelling of the membrane resembling that of acute otitis media or an acute exacerbation of the chronic middle ear suppuration. A similar case is reported by Hartmann (Politzer's Lehrbuch Ohrenheilkund., 1908, page 202.) The case seen by the writer has been reported by Ruttin (Sitzung der Oesterreich. Otol. Gesellschaft, Wein, Januar, 1908.)

A further possibility of confusion may be afforded by errors in the history, which is too often unreliable, a fact patent to everyone who has been obliged to take a great number of histories, especially in dispensary practice.

In this paper it will not be necessary to consider all of the diseased conditions of the nerve, since with most of them, including tumor, the similarity is not sufficient nor is the onset acute enough to permit confusion with labyrinth suppuration. We shall limit our discussion rather to neuritis of the Eighth Nerve; besides, a recognition of neuritis serves as a foundation for the recognition of the other diseased conditions of the nerve.

As in the case of the seventh, so in the case of the eighth nerve, neuritis may occur isolated or in combination with polyneuritis involving other cranial nerves. Oppenheim (Lehrbuch der Nerv. Krank., 1908) refers to a form of polyneuritis limited to the cranial nerves.

Combined seventh nerve paresis with herpes zoster (fifth nerve) has been observed and reported by Politzer (Lehrbuch der Ohrenheilk., 1908, page 611); Huet Fraser (Lancet I, page 18, 1904); G. G. Morrice (St. Barth. Hospital Reports, vol. xxxii, page 167, 1903); with sensibility disturbances of fifth nerve origin, by Huet (Soc. Neurol., 1904, Jan. 7th); by Donath, Spiller, Maurice, Mazurkiewitz and others.

Combined seventh nerve paresis with optic neuritis has been observed and reported by Wilfred Harris (Brit. Med. Jour., Jan., 1903); by Shumway (Jour. A. M. Assoc., vol. cliv, No. 6, page 63, 1905).

Combined eighth (acoustic) nerve paresis with fifth nerve paresis reported by Kaufman (Z. f. Ohrenheilk., 1897); Hammerschlag (A. f. Ohrenheilk., bd. xlv); Sarae (Z. f. Ohrenheilk., 1904); Lannois (Congress de Bordeaux, 1904); Gradenigo (Ann. de Mal de l'uereille, 1908).

Combined eighth nerve paresis with seventh nerve paresis has been reported by Rosenbach (Zentralblatt f. Nerv., 1887); Frankl Hochwart (der Menierische Symptom Complex, Wien, 1897) and others.

Combined eighth nerve paresis with fifth and seventh nerve paresis has been reported by Koerner (Munch. Med. Wochensch. nr. 1, seite 6, 1904).

Isolated paresis of the eighth nerve has been reported by Bing (Wien. Med. Wochenschr., 1880).

Isolated paresis of the cochlear branch of the eighth nerve has been reported by Gradenigo and Barnick (Arch. f. Ohrenheilk., bd. 28); Wittmaach (Zeitsch. f. Ohrenheilk., bd. 53); Hammerschlag, Kaufmann and others.

Isolated paresis of the vestibular branch of the eighth nerve has been reported by Ruttin (Sitz. der Oesterr. Otol. Gesell., Wien, Jan., 1908).

In looking over the literature I find that the cases of isolated neuritis of the eighth nerve are fewer than those of polyneuritis involving the eighth nerve. In the case of polyneuritis of the cranial nerves, the eighth nerve appears to be somewhat less vulnerable than the remaining nerves; however, with our increased knowledge of the functions and reactions of the vestibule and the vestibular nerve, our ability to recognize pathologic changes in the eighth nerve will be increased, and with it will come an increased number of these cases to be reported.

The causes of eighth nerve neuritis are generally the same as those of polyneuritis of the cranial nerves. Bezold divides the causes into three: (a) infectious; (b) constitutional; (c) toxic.

(a) Infectious. Polyneuritis during or after the infectious diseases is not uncommon. Of the infectious diseases causing neuritis we find prominently *tuberculosis,* especially the acute miliary form with fever; *typhoid fever,* especially those cases running very high temperature; *scarlet fever, influenza,* and *diphtheria.* The above fevers cause the acute forms of neuritis.

Besides these acute forms, we may have the more chronic primary gray atrophy of the nerve similar to that of the optic nerve in *metalues;* illustrated by the progressive deafness of tabetic patients (from 2 to 10 per cent.—Oppenheim). Relative to syphilis as a cause of neuritis, the writer observed two cases where the patients manifested hereditary syphilis with previous interstitial keratitis, and one in addition presented optic nerve atrophy.

(b) Constitutional causes are *leukemia, arteriosclerosis, carcinoma, diabetes, gout, malaria* and *cretinism.* Neuritis from all of these causes has been well authenticated and reported. These causes are prone to produce the isolated and unilateral forms of neuritis.

(c) Toxic. These are more frequently due to *quinine* and the *salicylates;* less frequently to *tobacco* and *alcohol,* and still less frequently to *lead, arsenic* and the inhalation of *carbon dioxide.* These tend to produce a form of retrolabyrinthine neuritis similar to the retrobulbar neuritis and resembles it furthermore in its amenability to treatment. Oppenheim suggests that in the toxic forms, especially in those cases from alcohol, tobacco, lead and arsenic, there may be two factors producing the neuritis: the poison acting as a predisposing and the exposure to cold and dampness as exciting causes.

(d) Refrigeratory, or so-called rheumatic, was not included in the above classification of causes since it is not exactly known

what this cause is. In most of these cases there appears to be two factors: first, auto-intoxication, and secondly, exposure to draught of air; at least, this last mentioned is the most frequently assigned cause by the patient. On the other hand, Alexander (Arch. f. Psychiatrie, bd. 35, heft 3) after a careful study of the microscopic pathology, suggests that the so-called rheumatic neuritis is probably of infectious (bacterial) origin. The illustrated plates which he produces would suggest such an origin; however, he was unable to prove the presence of micro-organisms with the Gram and methyline-blue stains which he used. There are, besides, two clinical facts which further support the theory of infectious origin for the so-called rheumatic polyneuritis; first, the usual presence of slight fever; secondly, the finding, although rare, of two members of the same family simultaneously affected with Bell's palsy.

PATHOLOGY.

Evidently the pathology is not the same in all forms of eighth nerve neuritis. The pathology in some of these forms is still unknown; however, in the acute cases thus far studied it was found that the nerve stem was infiltrated and swollen, while the nerve fibers were consequently destroyed and later replaced by proliferated connective tissue elements resulting eventually in a sclerosis of the nerve (Bezold, Lehrbuch der Ohrenheilk., 1906).

Hammerschlag claims that the pathology of eighth nerve neuritis is similar to that of seventh nerve neuritis with the exception that in the eighth nerve neuritis the end organ in the labyrinth is simultaneously affected, while Bezold, on the other hand, believes that the end organ is secondarily affected. In the case of neuritis from constitutional causes, at least in one variety—leukemia—Bezold found actual leukemic infiltration of the nerve stem.

The neuritis of toxic origin suggests a pathology similar to that of the retrobulbar neuritis; furthermore it behaves clinically the same, it is generally bilateral, and the prognosis is as favorable in the one as in the other under proper treatment; besides the two nerves, II and VIII, may be simultaneously affected by the same toxin.

In the so-called rheumatic form the pathology would suggest an infectious origin, but up to the present time the presence of the micro-organisms have not been demonstrated.

The post-syphilitic and the hereditary syphilitic neuritis of the eighth nerve from the clinical standpoint would suggest two separate path-

ologic processes; the former, an acute neuritis, and the latter rather that of a primary gray atrophy. This suggestion receives further support by the similar findings and prognoses of these two forms of changes found in the optic nerve from the same causes.

PROGNOSIS.

The prognosis depends in a measure upon the cause. In the post-infectious neuritis the prognosis is generally favorable, as in the case of neuritis of the other cranial nerves from the same cause. The prognosis in the constitutional is generally less favorable, and is in proportion to the prognosis of the primary condition behind it. The prognosis of the toxic forms is the most favorable under suitable treatment and by stopping the continuance of the poison which brought about the condition.

SYMPTOMATOLOGY.

In the cases of atrophy following neuritis the symptoms and signs, with the exception of the otoscopic findings, are the same as those of late labyrinth suppuration. Since however in atrophy the symptoms and signs are those of atrophy and not those of neuritis, we shall not consider them further in this paper. The object of this paper is rather to discuss the symptoms and signs of *acute* neuritis of the eighth nerve and its differentiation from *acute* labyrinth suppuration.

The symptomatology of acute neuritis depends upon the cause and extent of the process. Although neuritis of the eighth nerve occurs about as often unilateral as bilateral, we shall, in order to bring out more clearly the symptoms, refer especially to the unilateral form. In the cases of bilateral neuritis it also frequently happens that one side is much less affected than the other, and in this way, as in the case of double optic neuritis, the pathologic changes of the less affected nerve frequently escape detection and the nerve is incorrectly pronounced normal. In all cases of unilateral eighth nerve neuritis more careful attention to the other nerve will frequently enable us to avoid such mistakes and oversights.

Let us consider, first, the typical symptoms and signs of complete unilateral neuritis of the eighth nerve, and later we shall see that the more the symptoms and signs deviate from the typical the easier the differential diagnosis is.

The following symptoms and signs of acute eighth nerve neuritis are more or less abrupt in onset, with as a rule some fever and malaise

which may be more or less obscured by the more intense focal symptoms.

These are:—

1—Tinnitus or Subjective Noises. These in most cases are quite pronounced. The more acute the onset and the more complete the involvement, the less pronounced and the shorter the duration of this symptom. Theoretically, every attack of eighth nerve neuritis should present this symptom; however, it may be somewhat obscured by the more intense vertigo and thereby escape observation in a small minority of cases. In some cases the tinnitus continues in spite of complete deafness; two of such cases having been reported with galvanic reaction of the acoustic nerve by Dr. A. V. Mackenzie (Wein. Klin. Wochenschr., 1908, no. 11). In these cases tinnitus continues until complete destruction (degeneration) of the nerve takes place.

2—Deafness, complete, if the neuritis is complete. This follows quickly upon tinnitus in the acute cases. There is a loss of perception for all tones and noises upon the affected side, demonstrated by the small a_1 fork (Bezold) and the three meter speaking tube, alluded to in previous papers. Bone conduction is very much shortened, Rinné is negative with no air conduction, Weber to the opposite side, the acumeter and Galton's whistle are not heard upon the affected side; however, the absolute unreliability of these last two mentioned tests for one-sided deafness is well known.

3—Vertigo. The vertigo is most intense and is associated with nausea and vomiting. The patient suffers typical (Drehschwindel) turning vertigo, *i. e.*, he suffers the sensation as though the room were tumbling about him in the frontal plane or else the sensation as though he himself were falling in the frontal plane (laterally). This vertigo lasts several days or perhaps a week, depending upon the extent and intensity of the process. After the acute condition passes over the vertigo diminishes perceptibly and after a few weeks disappears entirely in those cases where the neuritis subsides and resolution takes place. Slight attacks of vertigo of brief duration may however continue in those unfavorable cases which are followed by atrophy.

4—Equilibrium disturbance. This is found constantly in all cases of neuritis, and is in direct proportion to the intensity and extent of the neuritis. At the beginning of the neuritis, the patient when attempting to stand falls toward the diseased side (typical reaction fall-

ing. This falling is not dependent upon the false impressions which are obtained through the *vision,* for, on the contrary, the tendency to fall is increased when the eyes are closed. The disturbances of equilibrium are so marked during the first few days that the patient is compelled to lie down for safety. This character of equilibrium disturbance is dependent upon and diminishes with the vertigo and eventually disappears entirely, being replaced by a late form of equilibrium disturbance in those cases of neuritis followed by atrophy. This late form of equilibrium disturbance is also found in late labyrinth suppuration. On the other hand, complete recovery from all equilibrium disturbances occurs where complete recovery from the neuritis takes place. It is not necessary to go further into the details of the character of the equilibrium disturbances since they are otherwise the same as those found in labyrinth suppuration.

5—Spontaneous rotatory nystagmus to the sound side. This nystagmus is very pronounced and is due to the overbalance of the normal tonus of the opposite (sound) nerve already mentioned in the first two papers upon labyrinth suppuration.

The nystagmus is furthermore rhythmic, the axial excursions of the eye balls are from three to four mm. in length at the corneal limbus. The nystagmus continues for a longer period than the vertigo, probably because the centers have become more or less adapted to the changed condition. In those cases of neuritis which terminate with resolution, the nystagmus disappears entirely or may even reverse its direction (to the diseased side): whereas in those cases of neuritis followed by atrophy, the nystagmus continues for months and eventually disappears, according to most authors. From a careful study of many cases however I doubt that the nystagmus to the well side ever entirely disappears, for in my experience I have never found it so, even in cases where the destruction has been of several years' standing.

6—Negative caloric reactibility of the nonacoustic labyrinth as found in labyrinth suppuration. No amount of cold or hot water syringed into the external canal produces any effect upon the existing spontaneous nystagmus. This negative reactibility remains pemanently so in those cases followed by atrophy, but not so in those cases of neuritis which result in recovery. In this latter instance the reactibility again becomes positive as before the attack.

7—Shortened duration of the after-nystagmus.

This sign belongs to any destructive lesion of the labyrinth, the nerve or Deiter's nucleus. In all of these conditions the duration of the after-nystagmus to the affected side is approximately one-half the duration to the sound side, so that an after-nystagmus of less than twelve seconds to the diseased side combined with an after-nystagmus of more than twenty-four seconds' duration to the sound side would indicate a loss of function upon the diseased side, and the after-nystagmus of twelve seconds' duration to the diseased side is purely a manifestation of a reaction from the well side. For a further explanation of these facts, the reader is referred to the second paper upon labyrinth suppuration. In cases of atrophy following neuritis, the shortened duration of the after-nystagmus to the diseased side remains permanently so, while in the cases resulting in recovery the duration of the after-nystagmus to the diseased side becomes as long as or even longer than that to the sound side.

8—Negative galvanic reactibility of the affected nerve. This sign is more or less present in both labyrinth suppuration and neuritis; however there is a difference between the reactions in these two processes, and I believe this difference affords us one of the best objective means for the differential diagnosis. For this reason, and furthermore since it was promised in the former papers, I purpose here to outline somewhat more in detail the facts concerning the galvanic reactions of the labyrinth and the nerve. These facts have been obtained by a series of examinations upon normal people, those with one-sided labyrinth affections (both irritative and destructive) and deaf mutes. For further details see Arch. f. Ohrenheilk., 1908.

Briefly, the *method of examination* was with an apparatus or wall plate fitted with three essentials: 1—an accurate galvanometer; 2—a switch to control the current, and 3—a reversing switch by which one can reverse the polarity without changing the electrodes. The two electrodes are: one large flat one to be appplied preferably to the opposite hand, and the other, a small ball electrode to be applied to the region of the ear; the latter is adjusted to a handle with an interrupter for making and breaking the current. In making the examination an assistant is required to control the current switch and to observe the milliamperemeter, while the examiner observes the eye movements holding the upper lid up with the thumb.

Both electrodes are covered with gauze, cotton or sponge, and are

kept well moistened with warm salt solution. The more saturated the electrodes are kept, the less the resistance and better the patient tolerates the examination. To aid in keeping the electrodes well moistened, I also have the skin of the hand and the region of the ear well moistened with the same salt solution.

As previously stated, my examinations included all varieties of cases and normal people. With normal people, I found the reaction from the static labyrinth to be more prompt than from the acoustic labyrinth; normally, from three to six milliamperes were required to produce positive reactions, *objectively* manifested by rotatory nystagmus, while from two to four milliamperes more were required to produce *subjective* vertigo. The difference in the strength of currents necessary to produce the objective and the subjective reactions explains the discrepancy in the figures obtained by myself and those of the earlier investigators who recorded only the subjective vertigo and did not look for the objective nystagmus. In all my figures I have noted the objective reaction (rotatory nystagmus) and have ignored the more crude and less reliable subjective reaction.

The examinations were all made with the patient looking straight ahead at a distant object, thereby preventing all inhibition of the vestibular reflex which might otherwise occur; besides, observations thus made are more accurate since in this position we avoid tremors of the eye muscles which might occur if the patient was to look in any other direction.

The results of my examinations show:

First—That the nystagmus is to the side of the kathode; second—away from the anode; third—that the kathodal reaction balances that of the anodal reaction; and fourth—with the same electrodes used, the two sides balance with each other.

Any imbalance indicates a pathologic condition of one or the other labyrinth or nerve.

1—When the kathodal reaction of one side is more prompt than the kathodal reaction of the opposite side, it indicates either a pathologic irritation of that side, or a pathologic destruction of the opposite side (labyrinth or nerve).

2—When the anodal reaction of one side is more prompt that the anodal reaction of the opposite side, it indicates a destructive lesion of that side or an irritative lesion of the opposite side (labyrinth).

3—When the kathodal reaction of one side is more prompt than the

anodal reaction of the same side, it indicates either an irritative lesion of that side or a destructive lesion of the opposite side (labyrinth or nerve).

The above findings alone are not sufficient for us to differentiate between these two processes (irritative lesion of one side or destructive lesion of the opposite side). The differentiation is accomplished by a comparison of the figures with those of the normal. Remembering that the normal reaction (nystagmus) is obtained with from three to six milliamperes with either electrode it follows that a reaction which requires more than six milliamperes indicates a destructive lesion, and a reaction requiring less than three milliamperes indicates an irritative lesion. Furthermore, if normally, four ma, with the kathode to the right ear will produce nystagmus to the right side, then four ma. with the kathode to the left ear will produce nystagmus to the left side in the same patient. The sum of these two reactions is eight ma. Again, if four ma. with the kathode to the right ear will produce nystagmus to the right side, then four ma. with the anode to the right ear will produce nystagmus to the left side; the sum of these two reactions is likewise eight ma. In the case of irritative lesion, this sum is proportionately less than eight ma. and in the case of destruction, proportionately more than eight ma. For instance in case of an irritative lesion of the right ear, we find most typically the reaction to be approximately as follows:

Right Side { Kathode 1 ma. rotatory nystagmus to the right side.*
Anode, 4 ma. rotatory nystagmus to the left side.

Left Side { Kathode, 4 ma. rotatory nystagmus to the left side.
Anode, 1 ma. rotatory nystagmus to the right side.

In case of destructive lesion of the right labyrinth, we find most typically the reaction to be as follows:

Right Side { Kathode, 8 or more ma. rotatory nystagmus to the right.
Anode, 4 ma., rotatory nystagmus to the left side.

Left Side { Kathode, 4 ma., rotatory nystagmus to the left.
Anode, 8 or more ma., rotatory nystagmus to the right side.

These results correspond very closely to the results obtained upon the turning stool, namely:—in case of one-sided i r r i t a t i v e lesion the sum of the number of turnings required to produce reactions to the two sides is *less* than the sum of the number of turnings required to

*These figures are more or less variable, depending upon the extent of the irritation and the duration of the process.

produce the same reactions in normal people. In the case of one-sided destructive lesion the sum of the number of turnings required to produce reactions from the two sides is *more* than the sum of the number of turnings required to produce the same reactions in normal people. These differences are still greater in the double-sided than in the one-sided affection (irritative and destructive).

In case of doubt we have the other reactions (caloric and turning) to help us to differentiate between these two affections.

From my experiments with the Galvanic reactions, I was enabled to conclude that the Galvanic nystagmus is produced by Galvanic irritation of both the end organ in the ampullæ and the vestibular nerve. In the case of labyrinth suppuration the nerve remains more or less intact for a considerable period of time (six to eight weeks) during which, though the Galvanic reaction is diminished, it is not entirely absent upon the affected side. This, so to speak, *rest* of Galvanic irritability upon the affected side must belong to the nerve *alone,* since the end organs in the ampullæ have been entirely destroyed by the suppuration. This fact was partly pointed out first by Neumann, who succeeded in producing, experimentally, Galvanic nystagmus by irritation of the exposed stump of the vestibular nerve in the wound cavity after labyrinth operation.

Later I was able to corroborate his results by the use of other methods.

In cases of acute labyrinth suppuration where the nerve is still intact, the results of my investigations showed that the Galvanic reactions with the kathode to the diseased ear required from eight to ten ma.; whereas, in case of severe neuritis or atrophy of the nerve I was not able to obtain the slightest evidence of a reaction with as much as sixteen, eighteen and twenty ma. (the strongest currents the patients could comfortably tolerate). The results were correspondingly the same when the anode was used, remembering of course that the nystagmus should be in the opposite direction to that produced by the kathode.

In the case of acute labyrinth suppuration where the nerve is still intact a sufficient amount of current with the anode will suppress the normal tonus of the intact nerve, whereas in the case of neuritis or atrophy where there is no normal tonus in the nerve, the anode, with no matter how great a current, produces no reaction.

I will not take up the space here to go into the details of the opening

and closing nystagmi, concerning which the reader is referred to a paper by the author (Klinische Studien ueber die Funktionsprüfung des Labyrinthes mittels des galvanischen Stromes, Archiv. f. Orhenheilk, Bd. 78. 1908). I will merely mention here a few of the more essential facts. (1) In normal people with the kathode to the ear, upon closing the current there is a nystagmus to that side; (2) upon breaking the current, there is a nystagmus to the opposite side of equal intensity to the closing nystagmus; (3) with the anode to the ear, upon closing the current there is a nystagmus to the opposite side; and (4) upon breaking the current, there is a nystagmus to the same side of equal intensity to the closing nystagmus; therefore, K C N = K O N and A C N = A O N. Deviations from these normal conditions indicate pathologic conditions. For illustration: in case of right-sided labyrinth suppuration, we have:

Right Side	K C N < K O N A C N > A O N	Left Side	K C N > K O N A C N < A O N

In the case of complete neuritis or atrophy of the nerve of the right side and in case of secondary degeneration following labyrinth suppuration we have:

Right Side	K C N and K O N negative A C N and A O N negative	Left Side	K C N > K O N A C N < A O N

In considering the differential diagnosis of labyrinth suppuration from neuritis, the history and the otoscopic findings afford us great aid. In labyrinth suppuration we have the positive history and otoscopic findings of middle ear suppuration, while in neuritis these are negative excepting in the rarest cases, where middle ear suppuration may be coincidently present. On the contrary, in the case of neuritis we are generally able to obtain a history pointing to one of the previously mentioned causes (infectious, constitutional or toxic).

In case of neuritis *complete* involvement of all branches of the eighth nerve is the exception rather than the rule. It frequently happens that the cochlear branch is more involved than the vestibular branch, and vice versa.

In neuritis it frequently happens that the involvement of the eighth nerve is but one of the manifestations of a polyneuritis; in which cases we are often able to detect pareses, (though often but slight), of one or more of the other cranial nerves.

In case of neuritis the affection is frequently bilateral, as elsewhere pointed out. The bilateral form occurs especially frequently from toxic

causes. A bilateral aeffction points decidedly to neuritis rather than to labyrinth suppuration.

From the foregoing we see that the greatest difficulty to be met with in the differential diagnosis occurs in the case of unilateral isolated complete neuritis of the eighth nerve with complete loss of both acoustic and static functions; fortunately, these are the rarest cases we meet, and in these cases the Galvanic reactions (see above) should determine the diagnosis.

In closing the subject, let us summarize briefly the more important differential symptoms and signs of these two processes.

LABYRINTH SUPPURATION.	NEURITIS OF EIGHTH NERVE.
1—*Positive* history of middle ear suppuration, together with the history of previous attacks of vertigo from labyrinth hyperæmia or labyrinthitis serosa.	1—*Negative* history of middle ear suppuration; but on the contrary history of infectious disease, or some toxic poisoning (quinine, salicylates, alcohol, tobacco, etc.); or exposure to cold and moisture.
2—Otoscopic findings characteristic for middle ear suppuration (secretion, perforation, polyps, cholesteatoma, etc.).	2—Otoscopic findings negative except in cases of herpes zoster involving the membrana tympani (see above), but in these cases we are usually able to detect the herpes blebs elsewhere in the region of the external ear and frequently on the auricle.
3 — Tinnitus and subjective noises *negative.*	3 — Tinnitus and subjective noises usually *positive,* and they last for indefinite periods of time from few hours to several weeks; in rare cases, followed by atrophy, even longer.
4—Deafness is *absolute.*	4—Deafness may be *absolute* or *partial,* and the type of this deafness is characteristic of that found in diseases of the perceiving apparatus.

LABYRINTH SUPPURATION—*Continued.*	NEURITIS OF EIGHTH NERVE.—*Continued.*
5—*Intense* vertigo lasting from three to five days, less pronounced for a few weeks longer during rapid movements of the head.	5—*Intense* vertigo lasting as a rule longer than in the case of labyrinth suppuration.
6—Equilibrium disturbances of the so-called vestibular type.	6—Same as in labyrinth suppuration.
7—Spontaneous rotatory nystagmus to the sound side.	7—Same as in labyrinth suppuration.
8—Negative caloric irritability of the static labyrinth.	8—Same as in labyrinth suppuration.
9—Shortened duration of the after-nystagmus to the diseased side.	9—Same as in labyrinth suppuration.
10—Galvanic reactions in recent cases only show the following average findings:	10—Galvanic reactions show the following average findings:
Right Side: Kathode, 7 ma., rot. nys. to r.	Right Side: Kathode, 18 ma., no reaction.
Right Side: Anode, 3 ma., rot. nys. to l.	Right Side: Anode, 18 ma., no reaction.
Left Side: Kathode, 3 ma., rot. nys. to l.	Left Side: Kathode, 3 ma., rot. nys. to l.
Left Side: Anode, 7 ma., rot. nys. to r.	Left Side: Anode, 18 ma., no reaction.
11—Paresis of other cranial nerves the *exception*, and then only the seventh nerve.	11—Paresis of other cranial nerves, more frequently the II, V, VII, XII; however, any of the others may be involved.
12—*Unilateral* involvement.	12—Frequently *bilateral* involvement.
13—*Permanent loss* of both acoustic and static functions.	13—In the majority of cases the prognosis is *favorable* with ultimate *recovery* of both acoustic and static functions.

PROGNOSIS AND TREATMENT OF LABYRINTH SUPPURATION

GREATER attention has been paid to the operative treatment of labyrinthine infections than to their diagnosis and differentiations. Originally the determination of the operative procedure upon the labyrinth was decided by the findings at the time of the radical mastoid operation. This form of practice might have done well enough for the past but not for the present.

Since the adoption of exact examination methods the normal physiologic functions of the labyrinth have been ascertained and at present we are able to determine, from variations in or by absence of these functions, pathologic conditions of the labyrinth. As a result, the best operators are adopting the plan of determining the exact condition of the labyrinth before performing *any* operation upon the ear.

It must not be forgotten that the best results are obtained not by the ability to operate alone but by the combination of this ability with a knowledge of the diagnosis; I might go further and say that the prognosis and treatment are entirely dependent upon the diagnosis.

The primary object of many operators has been to perform the labyrinth operations without a sufficient knowledge of the diagnosis and indications. This condition has been fruitful however in bringing about at least one result, namely, the perfection of the operations and a high development of surgical technique.

Many excellent papers have been written upon the surgical treatment of labyrinthine infections, including the recent one by Richards, of New York (*Laryngoscope,* Oct., 1907), and the still more recent one by Albert Jansen (*Annals of Otol., Rhin. and Laryng.,* June, 1908), however both authors in their respective papers show a lack of knowledge of the diagnosis and indications for treatment. Had they given more attention to the subject of diagnosis and the methods of examination, or had they familiarized themselves with the work of Barany (Phys. und Path. des Bogeng. Appar. beim Menchen), these men would have made fewer misstatements concerning the diagnosis, prognosis and indications for treatment.

Is it any wonder that Jansen's postoperative mortality (*Annals of O., R. and L.,* 1908, page 367), 25 per cent., should have been so high

as compared with Hinsberg's, 4 per cent. (furnished by Freytag, *Z. f. O.*, Bd. 51, page 341)? The explanation is that Hinsberg understood the diagnosis and operated when operation was indicated, while Jansen, who did not understand the diagnosis as well, operated hit or miss style, with the result that many labyrinths were sacrificed and lives risked if not lost that might have been saved by more conservative treatment.

After having carefully read Jansen's paper upon the "Treatment of Infective Labyrinthitis," I wish to protest forcibly against his misstatements concerning the diagnosis and indications for operation, because of the great danger of misleading some—fortunately not all—of the American otologists who may be inclined to accept his statements as authoritative.

Had Jansen familiarized himself more with what Barany and Neumann had done, rather than attempt to take from them the credit they deserve, he certainly would have known more about the diagnosis of labyrinthitis and the indications for operation than he manifested in his jumble of misstatements. On the other hand, Jansen deserves great credit for having inaugurated and helped to perfect the labyrinth operations.

In considering the prognosis and treatment of labyrinth suppuration it will be necessary at the same time to consider the prognosis and treatment of the other pathologic conditions of the labyrinth. These conditions will be considered separately.

I. Hyperemia of the Labyrinth. Passive hyperemia will not be considered here since it does not occur as a result of acute or chronic middle ear suppuration; while on the other hand, active hyperemia does (see former paper upon differential diagnosis).

The prognosis in active hyperemia of acute middle ear inflammation or suppuration is generally favorable, the hyperemia terminating in resolution. In exceptionally rare cases the hyperemia is but the initial stage of suppuration; in this latter instance the prognosis is generally unfavorable to the functions and to life, death occurring in from one to a few days from a suppurative meningitis due as a rule to very virulent streptococcic infection.

The treatment of hyperemia of the labyrinth with acute middle ear inflammation should be the prompt evacuation of the middle ear by a free paracentesis or the enlargement of a too small perforation, the application of drainage gauze into the external canal and moist heat,

absolute rest in bed and the administration of the indicated remedy. In the event of a serious turn in those rare cases mentioned with beginning meningeal symptoms the labyrinth operation should be performed early.

The prognosis in the hyperemia of chronic middle ear inflammation is even more favorable than in those cases occurring with acute middle ear inflammation. Since here we have generally a perforation of the tympanic membrane of considerable size we are denied the opportunity of making or enlarging the perforation. In these cases there is usually an acute exacerbation of the chronic middle ear suppuration or retention of secretion in one of the accessory cavities with adhesive bands, polyps or cholesteatoma.

The treatment should be directed toward the cause; in the event of an acute exascerbation of the chronic middle ear suppuration conservative treatment with rest in bed is generally sufficient. In the case of retention with polyps or cholesteatoma, these alone would give an indication for the radical operation, but not during the attack if it can be postponed for fear of precipitating an actual inflammation of the labyrinth; in these cases it would be better to operate between the attacks. In rare cases the hyperemia, as in the case of hyperemia of acute inflammation of the middle ear, might be the preliminary stage of a labyrinthitis, in which event the case must be treated accordingly.

II. Acute Diffuse Serous Labyrinthitis. Since the differential diagnosis of this condition from labyrinth suppuration is somewhat difficult; and furthermore since no exact statistics have been made, we are unable to say what per cent. of serous labyrinthitis recovers spontaneously and what per cent. terminates either in acute labyrinth suppuration or chronic labyrinthitis with new formed connective tissue and destroyed end organs. Fortunately however the prognosis in many of the cases, under proper treatment, is favorable both to function and to life.

The treatment during the attack should be both conservative and expectant; it is especially in these cases that waiting is profitable. There has been a tendency to commit one of two errors; either to do the radical mastoid operation immediately, with the result that the serous labyrinthitis develops into a suppurative labyrinthitis, or to operate the case as one of suppurative labyrinthitis with the result of permanent destruction of the functions. It would be far better after having first determined the diagnosis of serous labyrinthitis (see paper on

differential diagnosis) to put the patient to bed, insert drainage gauze into the canal, one end of which is introduced into the middle ear cavity, changing the gauze as often as necessary, and administer the indicated internal remedy. Such treatment will in the majority of cases result in recovery.

It is a mistake to attempt any manipulation or instrumentation of the middle ear during an attack of serous labyrinthitis. In order to prevent a recurrence or a subsequent attack of suppurative labyrinthitis we should immediately, after the attack of serous labyrinthitis has passed over, perform the radical mastoid operation and eliminate all infecting foci within the middle ear spaces.

III. Circumscribed Irritative Lesion of the Labyrinth. The prognosis in this condition is even more favorable under proper treatment than that of serous labyrinthitis, and the same remarks concerning the prognosis and treatment of diffuse serous labyrinthitis apply to this condition.

IV. Circumscribed Destructive Lesion of the Labyrinth. Since circumscribed destructive lesions of the membranous labyrinth are usually combined with similar changes of the bony labyrinth (labyrinth capsule) we shall speak of them together. Circumscribed destructive lesions of the membranous labyrinth are usually in the form of suppurations or granulations with surrounding zones of inflammatory reaction limiting the destructive pathologic changes to the circumscribed area. Later, in the surrounding zone of inflammation, adhesions are formed which serve as a barrier to the spreading of the infection.

The changes in the labyrinth capsule are in the form of fistulæ, erosions, caries and necrosis.

The prognosis is dependent upon the location. When the lesion is in the region of the semicircular canals (more often horizontal) the prognosis is more favorable than when in the region of the oval window or promontory, since in the former instance the canals being narrow, lumened tubes the adhesions can better shut off the infection from spreading than in the case of the vestibule or cochlea, where the labyrinth opens into wider spaces.

The prognosis too is dependent upon the treatment. The circumscribed destructive lesions of the labyrinth are usually slower processes than the diffuse serous or suppurative forms, as a result we have more time to consider our cases and the form of treatment to be applied In

the event of neglect of treatment there is the constant danger of slow or rapid progress of the circumscribed destructive process with the result eventually of an acute diffuse suppurative labyrinthitis. Realizing the dangers of a circumscribed destructive lesion, we should institute our treatment as soon as the diagnosis is determined.

The treatment consists of the radical mastoid operation and the exposure of the lateral labyrinth wall to full inspection. We should then eliminate, as thoroly as possible, all cholesteatomatous masses, granulations, necrotic bone, etc., using every precaution not to stir up the condition in the labyrinth for fear of breaking the adhesions, which may have formed, and thus favor an extension of the suppuration. This can be more readily accomplished in the case of a circumscribed destructive lesion of the horizontal semicircular canal than in a case of lesion about the oval or round windows or the promontory. In the latter cases (circumscribed destructive lesion about the windows or promontory) the danger of the conservative operation is too great, besides since the hearing is almost if not entirely destroyed we have not the same incentive to try the conservative operation. In these cases one of the more radical labyrinth operations should be performed.

V. Perilabyrinthitis with partial or complete sequestration of the labyrinth capsule.

The prognosis depends (1) upon the extent and acuteness of the process; (2) upon the nature of the predisposing cause (tuberculosis, diabetes, syphilis, scarlatina, etc); (3) upon the treatment, the promptness with which we get at it and the thoroness of the operation.

In all cases with complete sequestration of the labyrinth capsule there is facial paralysis. On the other hand, in cases of partial sequestration, especially when limited to the cochlea, the facial canal may escape involvement.

In some cases the entire labyrinth has been thrown off piece by piece thru the external canal without any untoward result; in other cases operative interference may be necessary. The prognosis to the functions is bad, but to life generally good. There is less tendency to intracranial complications in perilabyrinthitis than in labyrinth suppuration; however, complications do occur in exceptional cases, but in these the predisposing cause plays a role. In the cases of complete sequestration facial paralysis is complete and permanent, the only hope for future function lies in the performance of one of the nerve anastomoses operations.

The treatment should be at first prophylactic. In other words, all cases of mastoiditis in the course of acute or chronic middle ear suppuration should be operated promptly, following up the pus until normal tissue is reached. If this were done in every case, especially those showing early involvement of the facial nerve, there would be fewer cases of labyrinth sequestration. The simple mastoid operation in acute cases and the radical Kuester-Bergmann mastoid operation in chronic cases, including the removal of the mastoid tip, if done and done early will be sufficient to limit most cases of perilabyrinthitis. In the cases well advanced, after sequestration has taken place, the above mentioned operations are insufficient. In this event we must perform in addition to the mastoid operation an operation for the removal of the sequestered part or whole of the labyrinth, which will be found lying imbedded loosely in a dark granular mass of debris and pus, from which it can be removed easily with the anatomical forceps. This should be followed by thoro curettement of all diseased tissue.

Aside from the operative treatment it is well to direct sanitary and internal treatment toward the predisposing causes, to aid the after healing, which tends to be very slow in these cases.

VI. Suppurative Labyrinthitis (labyrinth suppuration). The prognosis in this condition would seem to vary with the skill of the diagnostician. It is almost impossible to compute the mortality, since in one clinic all cases of labyrinth infections are grouped together in one class, while in a second clinic the diagnoses are so loosely made that the figures become unreliable. For instance Jansen in his statistics includes cases which are positively *not* those of labyrinth suppuration, as does also Herzog. Herzog (Labyrintheiterung und Gehor, 1907) reports three cases (Nos. 3, 4, 5) among a total of seven in which the labyrinth suppuration was double-sided. Altho double-sided labyrinth suppuration is possible, that so high a percentage of such cases occurring in one man's experience seems highly improbable, especially when we recall the fact that such authorities as Hinsberg, Alexander, Neumann and Barany have failed to locate a single case during the last five years.

All authors agree, however, that the prognosis is generally unfavorable in untreated acute labyrinth suppuration, and much worse in cases maltreated; from which we may safely conclude that it is safer to leave the case to itself than to treat it improperly.

In the first place the prognosis for both acoustic and static functions

is *invariably* unfavorable; next the prognosis to life is rather unfavorable in the untreated cases, the infection finding its way into the brain—(1) thru the internal auditory canal, resulting in a circumscribed or diffuse serous or suppurative meningitis; (2) thru the aqueductus cochleae, resulting in a circumscribed or diffuse serous or suppurative leptomeningitis; (3) thru the aqueductus vestibuli, resulting either in an interdural (saccus empyema), deep extradural, subdural or cerebellar abscess. Altho the above results are the rule, exceptions do exist; in these exceptional cases two or more conditions may occur coincidentally thru two separate routes of invasion; for instance, from an acute infection of an old cholesteatoma simultaneous infection of the middle and posterior cranial fossæ with labyrinth suppuration may take place. In simple labyrinth suppuration the intra-cranial infection rarely takes place thru any but the preformed ways, for the reason that the infection tends to follow the line of least resistance.

The cases which terminate favorably are those where the suppuration limits itself to the labyrinth; the process of limitation of the suppuration taking place as a result of closure of the orifices of communication to the brain (internal auditory canal, aqueductus cochleae and aqueductus vestibuli) thru a serofibrinous inflammation, produced probably by the toxins of the infection and not the infection itself in a manner similar to the limitation of suppuration to the bulbus in case of panophthalmitis. This favorable result occurs in both instances (suppurative panotitis and suppurative panophthalmitis), especially when treatment has been expectant and conservative with absolute rest in bed and as little local interference as possible combined with the indicated internal remedy.

The indications for the labyrinth operation are not prompted by the labyrinth suppuration alone, but too by the meningeal irritation or inflammation and the primary middle ear suppuration.

Since it is dangerous to perform a radical operation in cases of labyrinth suppuration without at the same time opening or removing a sufficient part of the labyrinth for the purpose of drainage, we are compelled to do the complete operation. There are cases of very late labyrinth suppuration which have spent themselves within the labyrinth where a radical operation alone may be sufficient. However, I agree with Barany that such a procedure is rather dangerous, for the reason that we can never tell positively just how securely the suppurative process is shut in the labyrinth by the connective tissue adhesions about the

orifices, and if the adhesions are at all insecure the traumatism of the operation will rupture them with the result of a rapidly developing diffuse suppurative labyrinthitis.

The prognosis after the labyrinth operation is quite favorable, the mortality being from 4 per cent. (Hinsberg) to 25 per cent. (Jansen). The mortality in the Politzer and the Alexander clinics corresponds roughly with that of Hinsberg's.

The treatment of labyrinth suppuration may be conservative or radical. The conservative treatment is indicated first in those cases where in spite of the continuance of the symptoms and signs of lost acoustic and static functions, the headache, slight fever, the slight choking of the discs or overfilling of veins, gradually diminish; or in those late cases where from the lack of headache and fever and with normal fundus findings we have reason to believe that the diseased condition has become limited to the labyrinth.

When we have decided upon the conservative treatment it is best to put the patient to bed, use simple drainage gauze, and give the indicated internal remedy. It will be surprising what excellent results may be obtained in selected cases by such simple treatment. Personally however I am somewhat too timid to rely upon this treatment; knowing that a subsequent radical *must* be performed and the dangers it will invite, and furthermore believing that the labyrinth suppuration indicates a severe form of middle ear suppuration which may later give rise to other intracranial complications (sinus phlebitis, meningitis, extradural, cerebellar or temporal lobe abscess) I prefer to rely upon the radical treatment.

The radical treatment of labyrinth suppuration consists first in the performance of the Kuester-Bergmann radical operation, including complete evacuation of the mastoid process and removal of the tip. Good illumination is a prerequisite, best accomplished by the use of an Alexander electric head mirror (obtainable from Meyrowitz, of New York). From this step we have a choice of three methods for performing the operation upon the labyrinth; namely, the original method of Jansen, the method of Hinsberg and the method of Neumann.

JANSEN'S TYMPANIC OR SEMICIRCULAR CANAL METHOD.

Jansen has employed this first method since 1893. He begins by removing the upper wall of the anterior crus of the horizontal semicircu-

lar canal, allowing the lower wall to remain for the double purpose of serving as a protection against injury to the facial canal and for subsequent orientation. This trick is practiced also by Neumann, and both men claim the credit for having originated the idea. The posterior crus of the horizontal canal is then removed in its entirety. He next removes the bone below and backward from this part of the horizontal canal, taking care to avoid the facial canal. He then opens the posterior wall of the vestibule between the ampullar and smooth ends of the horizontal canal. To accomplish this with the least danger of injury to neighboring parts the chisel should be applied with the concavity externally when chiseling from the posterior crus into the vestibule and vice versa when chiseling from the anterior crus medianward, at the same time taking care to avoid the facial canal. He varies his method somewhat according to the pathologic changes found at the time of operation (the presence or absence of fistula).

After opening the posterior wall of the vestibule the anterior wall is sought. In determining the location of the anterior wall Jansen does not depend entirely upon the anterior crus of the horizontal canal but also upon the location of the oval window which he determines by the use of the bent probe introduced into the cavity of the vestibule.

Jansen feels that the exposure of the anterior wall is imperative in those cases of narrowing of the vestibule with new formation of bone. After determining the exact location of the lateral wall of the vestibule by thoro sounding with the probe within the cavity of the vestibule, he then removes it with great care with either a narrow chisel or burr. He prefers the use of the chisel when he wishes to ascertain a knowledge of the vestibule contents, otherwise he prefers the use of the burr. He advises the use of the burr too especially to preserve the facial canal and the inferior wall of the anterior crus of the horizontal canal. He advises us of the importance of reaching the deepest point in the vestibule, laying bare its floor. He next enlarges the cavity of the vestibule downward by exposing the lower orifice of the inferior semicircular canal and enlarges it backward by exposing the common orifice of the vertical canals. He advises the use of the burr in enlarging the cavity backward. He finally exposes the ampulla of the superior semicircular canal. Rarely does he follow up all of the canals. He cautions against too vigorous enlargement of the oval window backward on account of the danger of narrowing the bridge of bone containing the facial nerve to the extent that necrosis may follow.

Jansen claims that this operation is the safest and fraught with the least danger to life. He does not consider the operation upon the cochlea as always necessary; however, when he does deem it necessary he recommends the following method:

OPERATION UPON THE COCHLEA.

Jansen has modified his former method of complete removal of the cochlea to that of removal of the promontory and the exposure of the lower cochlear whorl only, at the same time avoiding injury to the modiolus. He has avoided injury to the modiolus since having lost at least one case by such injury with a probe. On the other hand, when the cochlea is transformed into a sequestrum lying in a bed of granulations and pus he effects its removal with the gouge.

THE HINSBERG METHOD.

The first step in this method is to perform the complete radical operation, including the removal of the posterior wall of the external canal downward to the level of the floor of the canal and the shaving down of the facial spur as far as possible without injury to the nerve, thus permitting the best possible inspection of the region of the oval window, the facial canal and the prominence of the horizontal semicircular canal. He next seeks the oval window. If the stapes is still there he removes it, then with the very small bone forceps of Bourquet introduced into the oval window he proceeds to remove the lateral vestibular wall to the round window and at the same time opens the lower cochlear whorl; he then lays the bone forceps aside and proceeds to remove the promontory anteriorly using the chisel, which he prefers to the burr since he can keep a clearer field, the burr producing too much bone dust and thereby obscures the field. He, like Jansen, is very careful not to work backward toward the facial. Hinsberg then introduces a bent probe, or better, the Bourquet facial nerve protector, into the enlarged opening, carrying it backward and slightly upward behind the facial until its tip reaches the anterior crus of the horizontal canal, which he then proceeds to remove with the Bourquet forceps. He completes his operation according to the findings, by opening or not the remaining canals.

Hinsberg like Jansen deviates from his routine course in case of fistula. In the event of previously long standing facial paralysis with cholesteatomatous invasion of the labyrinth, where oftimes the facial

canal has been eroded and the nerve more or less destroyed, he does not spare the facial canal but proceeds directly to remove it, trusting to a subsequent facial nerve anastomosis rather than to the dangers invited by a less radical procedure.

THE NEUMANN ENDOCRANIAL METHOD,

Is a modified and improved endocranial method of Jansen which he (Jansen) claims to have performed as early as 1895, in cases of deep extradural abscess. Jansen has since abandoned this method more or less because, as he claims, it is a more difficult and dangerous procedure than his socalled "tympanic method."

The operation as Neumann performs it consists first in performing the most complete radical operation, including a thoro shaving down of the facial spur, thoro removal of the inner mastoid wall and also the mastoid tip, free exposure of the posterior and middle skull fossæ. For this method of radical operation Neumann claims the advantages, first, of thoro inspection of the neighboring vital structures (middle and posterior fossæ) for any pathologic changes that may have clinically escaped our attention; second, of permitting the greatest possible room for the performance of the subsequent labyrinth operation; third, of more prompt healing after the operation, since the dura of both the middle and posterior fossa tends to prolapse somewhat into the wound cavity, thereby lessening its size.

The second part of the operation consists in complete removal of the semicircular canals, free opening of the vestibule and cochlea. This part of the operation is begun by removing the posterior wall of the pyramid with the chisel from behind, the posterior vertical canal is removed and the common orifice of the two vertical canals, then the posterior crus of the horizontal canal. The removal of the posterior wall of the pyramid is continued to the internal auditory canal. The danger of opening the subdural space (as Jansen fears) at the entrance of the ductus endolymphaticus is very slight indeed, since the saccus endolymphaticus lies between two layers of the dura (interdural space) and not below the dura. The anterior crus of the horizontal canal is left till later. The bone in the solid angle between the three canals is attacked next to gain an entrance into the posterior part of the vestibule, this is accomplished by using the smallest chisels (Alexander, number 6 or 7). After opening the vestibule at this point the opening is enlarged very carefully, often by using the chisel as a plane, removing the bone in thin

layers. The lateral vestibular wall is then thoroly removed and next the promontory, permitting the freest possible opening of the cochlea. At this stage a probe introduced into the anterior opening behind the facial canal should present itself thru the posterior opening. The bridge of bone containing the facial canal is well preserved and to aid in its preservation as much of the anterior crus of the horizontal canal as possible is allowed to remain until the final step. The final step consists in removing the entire upper wall of the horizontal canal to the ampulla. Neumann usually includes in his operation the removal of the lateral wall of the internal auditory canal. He does not fear to the same extent the dangers of injury to the facial canal, the carotid, the internal auditory canal and the bulbus jugularis as does Jansen, and I believe, too, that these dangers are over estimated and are really of less importance than the thoro removal of the diseased parts.

COMPARISON OF THE OPERATIONS.

The Jansen "tympanic" method has the following disadvantages:

1. If, as Jansen himself cautions against, the dura is accidentally exposed or injured at the time of the operation, the injury is so fine as to be invisible and escapes our notice; this makes the danger of complications following vastly greater than where the dura is well exposed or a larger incision of the dura has been made. The dangers of small invisible injuries to the dura have been pointed out long since by Koerner and Alexander.

2. In the event of slight or beginning intracranial complications (extradural granulations or abscess, serous meningitis, etc.) which may have escaped clinical diagnosis we are unable to inspect the parts at the time of operation.

3. In the event of intracranial complications following the labyrinth operation, they are much more difficult to get at, because of the presence of the extra bone and tissue we are compelled to remove, which would not exist had the endocranial method of operation been originally performed.

4. When the cochlea, which is always involved in acute diffuse labyrinth suppuration, is left unopened, as Jansen frequently does, the danger of a meningitis is quite as great as tho he had performed no operation at all. This undoubtedly accounts for his exceedingly high post operative mortality (25 per cent.).

Since advantages imply a comparison of one with another, I fail to

see any advantage of the Jansen operation over that of the Neumann modified endocranial method, or even the Hinsberg operation.

Disadvantages of the Hinsberg operation are much the same as those given for the Jansen; they are briefly:

1. Too little room to work, as in the Jansen tympanic method.

2. The bone forceps of Bourquet used to enlarge the oval window are apt to slip and injure the facial behind, or by using too much pressure the tip of the forceps may injure the carotid in front. At best the forceps are less under our control, obscure our field more and are generally clumsier than either the chisel or the burr.

3. Since Hinsberg's operation is a "tympanic" method like that of Jansen's, it is subject to the same general disadvantage (see above).

Hinsberg's method has at least one pronounced advantage over that of Jansen's in that Hinsberg includes the opening of both vestibule and the cochlea; hence his much lower post operative mortality (4 per cent. in 69 cases—Heine, Oper. am Ohr., page 118) than Jansen's (25 per cent.).

The Neumann method has no disadvantage compared with the Jansen or Hinsberg methods; on the other hand, it has many advantages—fewer however over the Hinsberg than over the Jansen method; they are:

1. The advantage of the greatest possible exposure of the field of operation to the view of the operator, with an opportunity of inspecting the dura of both fossæ and the sinus.

2. The advantage of the greatest possible room for the manipulation of instruments in the performance of the operation.

3. The advantage of increased accessibility to intracranial structures in the event of postoperative complication.

4. Quicker afterhealing than after either the Hinsberg or Jansen operation.

The advantages and disadvantages of the three methods of operating can be told furthermore mathematically by a comparison of the figures of the postoperative mortality. Out of 100 cases Jansen's postoperative mortality is 25 per cent. Out of 70 cases Hinsberg's mortality about 4 per cent. Out of the not yet computed number of cases operated since 1906 in the Politzer and Urbantschitsch clinic after the Neumann method a mortality of 0 per cent. One case, however, ended fatally as a result of an intercurrent attack of facial erysipelas (Barany, Phys. u. Path. des Bog. Ap., 1907, page 49).

It is claimed that figures do not lie, nor do they; but at the same time I will not go so far as to say that these differences in mortality were due entirely to the differences in the methods of operation employed but also to the difference in the ability of these men to make the diagnosis and select their cases from the indications.

In closing the subject I still insist that the ability to operate is of secondary importance to our ability to diagnose, and I would trust myself with labyrinthine infection in the hands of a diagnostician before I would the operator, providing the one knew nothing of the subject of the other.

DRESSINGS AND AFTER-TREATMENT.

At the completion of the labyrinth operation the wound cavity should be thoroly cleansed and washed out. In doing this some operators use hydrogen dioxide, others a weak formaldehyde solution (½ to 1 per cent.), and still others use both solutions, the hydrogen dioxid first followed by formalin; each of these solutions to remain in the wound cavity for a very brief period. The danger of hydrogen dioxid spreading the infection to neighboring parts is less feared today than formerly, and most surgeons doubt its possibility altogether. Whether the one or the other or both of these solutions have been used, they should be followed by copious quantities of sterile water.

After thoro cleansing of the wound cavity, as above described, a careful inspection should be made of the neighboring parts, especially of the sinus and dura, for other complications.

Normally the sinus should present a smooth, somewhat glistening surface, blue in color and normally distended. The dura should present a smooth glistening surface, of a pearly, faintly bluish white color; it should be very loosely adherent to the inner surface of the bone, from which it can be readily separated by a suitable instrument (dura proector); furthermore there should be neither bulging nor prolapse of the dura. Any deviation from the above appearances of sinus or dura indicates some pathologic condition.

Granulations on the sinus are recognized by the red or brownish discoloration with unevenness of the surface and the tendency to bleed. Occasionally there may be free pus found in association with these granulations, when the condition is spoken of as a perisinus abscess. These granulations, or perisinus abscess, may or may not be associated with sinus phlebitis and thrombosis but more often they are not. The

treatment consists of the thoro removal, preferably by bone forceps, of all overlying bone until normal sinus wall is reached, so that at the completion of our efforts the granulations or abscess should be completely surrounded by a zone of healthy tissue.

The subject of sinus thrombosis is so immense that the writer will not even attempt a brief outline of the subject at this time, but will refer those interested to the text books; to Koerner's work—"Erkrank des Hirns der Hirnhaute und der Blutleiter"—or to a recent paper by Alexander, translated by Dr. Geo. Davis, of New York (*Archives of Otology*, 1908).

Extradural granulations are recognized by the same general appearances as when found upon the sinus wall. These may also be associated with free pus, when we recognize the condition as an extradural abscess.

In every case of extradural abscess a careful inspection should be made for the purpose of determining the presence of a communicating fistula leading to a brain abscess. Where no fistula is found we should treat the extradural granulations or abscess after the manner of treatment for the same condition of the sinus described above. If on the other hand the presence of fistula has been determined it should be enlarged and the deeper brain abscess opened and drained. For this purpose the writer advises the use of a freshly sterilized, double edged, graduated brain knife. Puncture needles are to be condemned as too dangerous and unreliable instruments to be used in the brain or sinus.

Bulging or prolapse of the dura thru the openings which have been made in the skull fossa indicates, in a general way, an increase of intracranial pressure. A moderate degree of circumscribed bulging with discoloration of the dura (usually dark) indicates subdural abscess. The treatment consists of free opening of the abscess with a freshly sterilized knife. A moderate degree of prolapse of the dura indicates more frequently a serous leptomeningitis or meningoencephalitis, less frequently a brain abscess. The degree of prolapse in brain abscess is variable, depending upon the size and the stage of advancement of the abscess. In all suspected cases of brain abscess free paracentesis with the previously mentioned brain knife should be made, and it is surprising how frequently our suspicions will be verified. In all cases of prolapse, even of moderate degree, the dura should be freely incised, at least one large incision for the posterior and a second one for the middle fossa. In case of serous meningitis or meningoencephalitis the brain

substance prolapses thru the dural incision. If the prolapse is very marked and the convolutions flattened to any extent a further puncture of the lateral ventricle of that side is indicated.

A marked degree of prolapse indicates suppurative meningitis. In these cases the symptomatology will have aided us considerably in our diagnosis. Here the same incision as in the case of serous meningitis is indicated, together with puncture of the ventricle and lumbar puncture. When making the dural incisions a milky or purulent fluid escapes. With suppurative meningitis the prolapse of brain substance thru the dural incision acts more or less as a plug, interfering with the drainage of pus from the surrounding areas; for this reason some have attempted, with more or less success, the making of counter openings in the skull and dura at distant points.

In suppurative meningitis when not too far advanced this operative treatment has been fairly satisfactory and more is to be hoped for in the future.

DRESSINGS.

The materials required for the dressings are the same whether the operation for labyrinth suppuration alone has been performed or whether a more complex operation to include the treatment of some of the other complications. The only difference in these cases is in the manner of applying the dressings.

The materials needed are (a) freshly sterilized moist iodoform gauze, prepared in narrow strips (1 to 1½ inches wide) and of convenient lengths for handling. As a substitute for the iodoform gauze some operators have been using iodoform docht (the German for wick) prepared in small bundles of ten or a dozen strands each, the bundles held loosely correspond in size to the thickness of an infant's finger. For the wicks the advantages are claimed that they are easier to introduce, especially into small openings, are more readily removed and with less discomfort to the patient than iodoform gauze since a strand or two may be removed at a time if so desired.

(b) Additional strips of iodoform gauze, about 2 inches wide and of considerable length, but folded.

(c) Plain gauze, folded into 4 inch squares, of four or five thicknesses.

(d) Gauze bandage.

(e) Starch bandage or adhesive strips.

In case of labyrinth suppuration without other complications, the dressings are applied according to the following: After ligating the vessels and suturing the plastic flap or flaps, we introduce the moist freshly sterilized iodoform gauze or wick, first into the very bottom of the wound cavity, and then fill up the cavity partially. A second piece of the same material is then introduced into the cavity thru the external meatus; this second piece should be packed in tightly against the plastic flap in its new position. The entire cavity of the wound is then filled up to the level of the surface with the remnants of the posterior piece if the quantity is sufficient, or a third piece may be used for the purpose. Most operators prefer a fairly tight packing for the first dressing. Over the moist iodoform gauze or wick a few strips of the wider iodoform gauze is applied, and over this the plain gauze, using 8 or 10 squares. The first 4 or 5 plain gauzes should be unfolded and laid on loosely thus moulding the dressings better, they also tend to stick or adhere less when we come to remove them. Over all is applied a 2 inch gauze bandage (personally the writer prefers the Koerner method of bandaging), and finally a starch bandage or adhesive plaster is used to hold all in place.

It is never advisable to sew up the wound, partially or competely, after the labyrinth operation or where the cranial cavity has been opened. Secondary closure of the retroauricular opening may be done after a few weeks.

The only variation in the dressing of those cases associated with other complications is in the application of the dressings. In these cases care should be exercised to see that the moist iodoform gauze or wick is applied directly to the granulations or into the openings made in the dura in cases of meningitis or into the abscess cavity in cases of brain abscess.

After the operation the patient should be put to bed and kept there until the temperature reaches normal and the patient feels comfortable. Internal medication will be decided by the symptoms as they may arise.

The dressings need not be touched for from 6 to 10 days if the case does satisfactorily. After a few days a rise of temperature, pain or saturation of the dressings may call for a change of dressing.

A rise of temperature may indicate some acute infectious fever, tonsilitis, facial erysipelas or simple autointoxication from constipation or the extension of intracranial infection.

Slight pain may indicate nothing. More intense pain may indicate a

too tightly applied bandage or the spreading of infection; especially meningitis when the pain is associated with cerebral vomiting, choked disc, diminution in the pulse rate, etc.

Early saturation of the dressings with bloody colored watery fluid indicates escape of cerebrospinal fluid.

All of these conditions indicate early partial or complete redressing of the wound. On the other hand, if every thing goes along smoothly the first redressing may be made on or about the sixth day. At this time we should remove all that can be removed readily. In case the iodoform gauze immediately next the wound surface adheres firmly it is best not to remove it but to allow the adhering part to remain until the next redressing. Providing the wound shows healthy granulations the first redressings should be made considerably looser than the primary dressing.

Redressings should be made every day or two, according to the behavior of the wound and the amount of secretion. The greater the amount and the more purulent its character the oftener the redressing should be made.

In labyrinth suppuration healing takes place about as promptly or more promptly than after one of the radical operations; furthermore, after the third or fourth redressing less care need be exercised than in the case of the radical mastoid where the conservation of hearing becomes a factor in the after-treatment.

After the labyrinth operation the rapid filling up of the wound cavity with granulations resulting in obliteration of the middle ear spaces is not contraindicated as in other cases of ordinary chronic middle ear suppuration.

The secondary sutures for closure of the retroauricular opening may be performed after two or three weeks or when all acute symptoms have disappeared. Final healing takes place in from two to three months, when the patient may be discharged.

PHYSIOLOGY AND PATHOLOGY OF THE NON-ACOUSTIC OR, SO-CALLED, STATIC LABYRINTH

The Anatomy.

FOR the thorough understanding of the physiology of any part of the body one must have at least a fair knowledge of the anatomy (gross and microscopic) of that part, together with some knowledge of its relation to other parts. These facts are especially true with regard to the labyrinth, and for this reason the author will devote the entire first paper to this part of the subject. It is not intended that this paper should be a complete work upon the anatomy of the ear; on the contrary, only as much of the anatomy will be considered as is absolutely necessary for the understanding of the physiology and pathology and the reactions of the labyrinth under normal and pathologic conditions to be described in subsequent papers.

OSSEOUS LABYRINTH OR LABYRINTH CAPSULE.

The osseous labyrinth is formed of compact, densely lamellated bone, quite complex in shape as its name would indicate and located within the pyramid of the temporal bone. It is surrounded, with the exception of parts of its lateral surface, with diploetic or pneumatic bone. When of pneumatic bone, the cells form part of the mastoid cells, are lined with mucous membrane and are in direct communication with the mastoid antrum.

The character of the bone surrounding the osseous labyrinth is important, less from the physiologic than from the pathologic standpoint. The danger of labyrinth sequestration from an attack of mastoiditis is increased directly in proportion to the pneumatic and inversely to the diploetic character of the surrounding perilabyrinthine bone.

The osseous labyrinth is composed of three essential parts: (1) The cochlea; (2) The vestibule; (3) The three Semicircular Canals.

The **osseous cochlea** is a hollow tube, wound 2½ times about a conical shaped axis called the modiolus. The largest diameter of the tube is at the base and it tapers gradually toward and terminates in a blind end on the summit of the modiolus, known as the cupula cochleæ. The cochlear tube is partly divided into two parts by a spiral shaped

process of bone, called the lamina spiralis ossea, which projects from the modiolus and runs its entire length, terminating in a sickel shaped lip at the apex, called the hamulus. The lamina spiralis ossea incompletely separates the cochlea into a lower semicircular shaped tube, known as scala tympani, and an upper one, known as the scala vestibuli. The separation of the two scalæ is completed by the lamina spiralis membranacea (of the membranous labyrinth) excepting at the apex where the two scalæ communicate around the tip of the hamulus; this space of communication is known as the helicotrema. The scala vestibuli opens at the lower end into the cavity of the vestibule. The scala tympani ends somewhat beyond the fenestra rotunda. The fenestra rotunda is closed by the membrana secunda.

The modiolus is composed of spongeous bone and contains a fairly large spiral shaped canal which also terminates in a blind end at the apex. This canal accommodates the ganglion spirale cochleae. Another smaller spiral shaped canal within the modiolus accommodates the artery, ramus cochleae a branch from the arteria auditivæ internæ, and its subdivisions. The accompanying veins run in a separate canal. The base of the modiolus presents inwardly at a slight angle to the internal auditory foramen.

In the scala tympani of the lowest whorl of the osseous cochlea just anterior to the fenestra cochlea is a small opening, the apertura interna canaliculi cochleae, for the transmission of the perilymphatic duct (ductus perilymphaticus).

The osseous cochlea contains the pars inferior labyrinthi, which comprises the hearing organ.

The **vestibulum** is that part of the osseous labyrinth which is more or less sack shaped, lying between the cochlea anteriorly and the semicircular canals posteriorly, taking up much less space than either. On its upper and outer surface are two rounded prominences which lie just next to each other with a wedge shaped depression or incision between them; the anterior prominence is known as the recessus sphericus, a better name would be prominentia sphericus; the posterior is known as the recessus ellipticus. The depression between the two prominences is known as the crista vestibuli. As the names imply, the recessi and crista are strictly speaking correct only when viewed from the inner aspect. The recessus sphericus accommodates the sacculus and the recessus ellipticus the utriculus of the membranous labyrinth,

and the depression on the outer surface corresponds with the crista between these two recesses.

On the external surface of the vestibule, a short distance below the two prominences above mentioned, is a kidney shaped window, the fenestra vestibuli, the long axis of which is somewhat oblique from above and forward to below and backward; the concavity corresponds to the hilus of the kidney, is downward and forward. This window is closed by the stapes plate (basis stapedis). There is no anterior surface of the vestibule but that part corresponding to it opens into the lower cochlear whorl, so that the vestibule appears somewhat as a more or less rounded body resting upon but too large to fit into a cornucopia.

The anterior inferior part of the vestibule is represented by that part of the lowest cochlear whorl which is covered by the secondary membrane and slightly overlaps the floor of the vestibule; the part just posterior to this represents the floor of the vestibule.

Posteriorly the semicircular canals open into the vestibule through five openings; the three ampullar ends of the three canals, one separate smooth end for the external and one common smooth end for the two vertical canals.

From the median or inner side the prominences representing the recessus sphericus and ellipticus can be seen above, the common crus of the two vertical canals posteriorly, the ampullar end of the inferior semicircular canal inferiorly and a very narrow space in the center of all three in which a foramen—the apertura externa aqueductus vestibuli—is located which allows for the transmission of the ductus endolymphaticus.

The **semicircular canals** are three in number: (a) the *External*, Horizontal or Lateral; (b) the *Superior* or superior vertical; (c) the *Posterior*, Inferior, Inferior Vertical. These canals are more or less cylindrical shaped tubes bent in the form of semicircles or a trifle more than a semicircle, they are of almost uniform transverse dimensions, excepting at one end where each canal terminates in a somewhat spherical shaped swelling called the ampulla. Each canal therefore has two ends, the ampullar and the nonampullar or smooth end. The ampullæ of the three canals all open directly into the vestibule. The smooth end, crus simplex of the horizontal canal does likewise, but the smooth ends of the two vertical canals open into a common tube, the crus commune, and it in turn into the vestibule.

The locations of the ampullæ are more or less important. The ampulla of the horizontal canal is located at the anterior end of the crus ampullare just behind the facial canal, which separates it from the oval window on the lateral aspect of the vestibule; the ampulla of the superior canal is immediately above the ampulla of the horizontal semicircular canal on the upper posterior aspect of the lateral wall of the vestibule. The ampulla of the inferior semicircular canal is located just posteriorly and medianward to the inferior wall of the vestibule.

The nonampullar or smooth end of the horizontal semicircular canal opens into the vestibule at its posterior part.

The common orifice of the vertical canals opens into the vestibule at its posterior median part

The description of the facial canal and its course will not be given in this paper since it does not belong strictly to the subject.

That part of the osseous labyrinth which is exposed laterally forms the greater part of the inner wall of the middle ear, and is recognized from the tympanic side as the prominence of the horizontal semicircular canal (prominentia canalis semicircularis lateralis), part of the lateral vestibular wall and the promontorium corresponding to the superior, posterior and lateral part of the cochlea.

This exposure of the lateral wall of the labyrinth is of clinical importance in the making of the caloric test, as we shall learn later, and of pathologic importance since it offers especially vulnerable points for the infection of the labyrinth from middle ear suppuration.

In addition to the exposed parts just mentioned, we have two windows of the labyrinth exposed laterally, the fenestra vestibuli or ovalis and fenestra cochleæ or rotunda, which normally are sealed up, the former with the basis stapedis, the latter with the membrana secunda. These closed windows are of physiologic importance to the function of hearing, but under the destructive influence of middle ear suppuration they may become open gates for the spread of infection to the labyrinth.

The basis stapedis separates the tympanic cavity on the external side from the perilymphatic space in the vestibule on the median side. The membrana secunda separates the tympanic cavity on the external side from the perilymphatic space at the bottom of the scala tympani on the median side.

The fenestra vestibuli and the fenestra cochleae are both located at

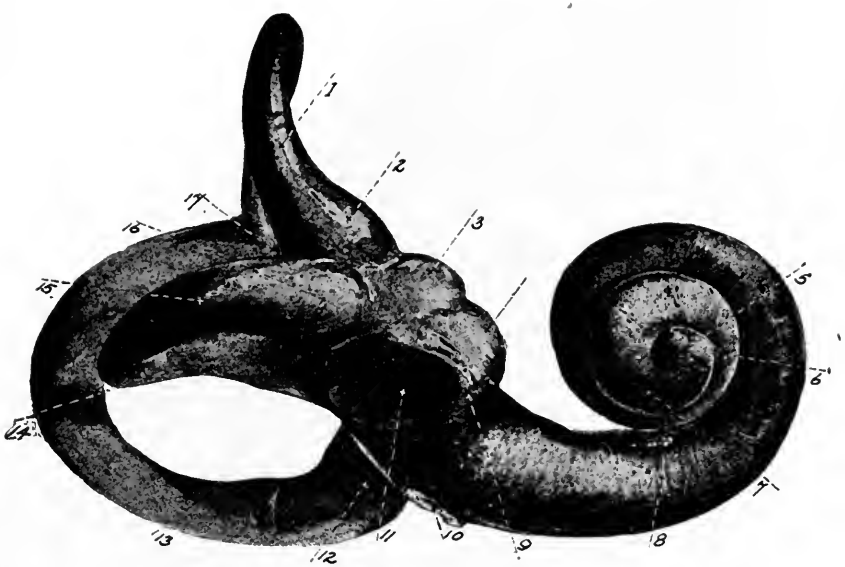


Figure 1.—A mould of the right osseous labyrinth (lateral view) after Spalteholz.

1—Canalis semicircularis superior. 2—Ampulla ossea superior. 3—Recessus ellipticus. 4—Recessus sphericus. 5—Cupula cochleae. 6—Middle cochlear whorl. 7—Upper cochlear whorl. 8—Lower cochlear whorl. 9—Location of crista vestibuli. 10—Fenestra cochleae. 11—Fenestra vestibuli. 12—Ampulla ossea posterior. 13—Canalis semicircularis posterior. 14—Crus simplex. 15—Crus ampullare. 16—Ampulla ossea lateralis. 17—Crus commune.

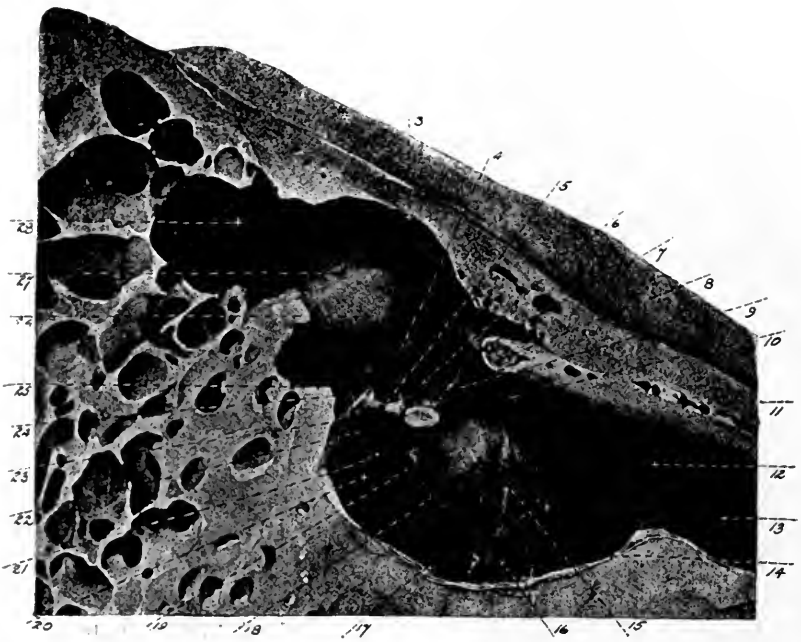


Figure 2.—Median wall of the right tympanic cavity of the adult human—after Spalteholz (enlarged 5 diameters).

1—Boundary between antrum tympanicum and recessus epitympanicus. 2—Tegmen tympani. 3—Paries tegmentalis. 4—Prominentia canalis facialis. 5—Tendo M. stapedii. 6—Plica stapedis. 7—Processus cochleariformis. 8—M. tensor tympani. 9—Stapes. 10—Paries labyrinthicus. 11—Septum canalis musculotubarii. 12—Ostium tympanicum tubæ. 13—Pars ossea tubæ. 14—Paries caroticus. 15—Plexus tympanicus (Jacobsoni) im sulcus promontorii. 16—Cellulæ tympanicæ. 17—Paries jugularis. 18—Promontorium. 19—Fossula fenestræ cochleae. 20—Subiculum promontorii. 21—Paries mastoideus. 22—Sinus tympani. 23—Eminentia pyramidalis. 24—Sinus posterior. 25—Fossa incudis. 26—Prominentia canalis semicircularis lateralis. 27—Recessus epitympanicus. 28—Antrum tympanicum.

the bottom of bony niches (see Fig. II). These niches serve normally as a protection against direct injury; on the other hand, during an attack of middle ear suppuration they serve as reservoirs for the retention of pus and as beds for granulations. Thus these niches while

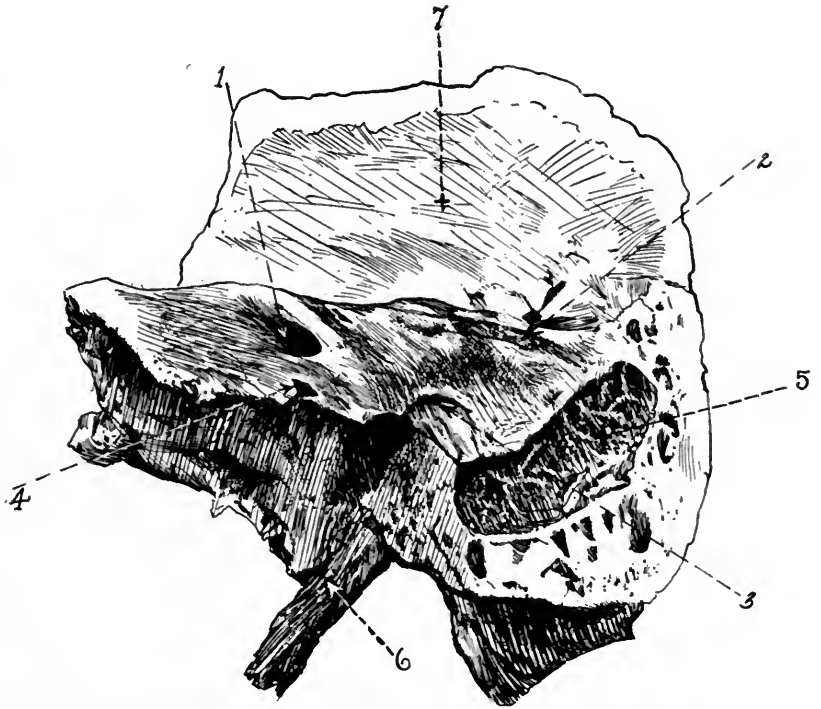


Figure 3.—The right temporal bone viewed from the posterior median and slightly inferior aspect.

1—Meatus acusticus internus. 2—Superior petrosal groove. 3—Aqueductus cochleae. 4—Aqueductus vestibuli. 5—Groove for the lateral sinus. 6—Processus styloideus. 7—Inner surface of the squamous portion.

diminishing the vulnerability of the labyrinth to traumatism increase it to infection.

The labyrinth capsule is perforated cranialwards by its three foraminae, two of which are represented on the posterior median surface of the temporal bone by the meatus acusticus internus and the aqueductus vestibuli; and the third, aqueductus cochleae, is directly below the meatus acusticus internus.

The **meatus acusticus internus** perforates the posterior median surface of the petrous bone at about one-third the distance from the tip in an oblique direction outward and slightly backward, so that the angle

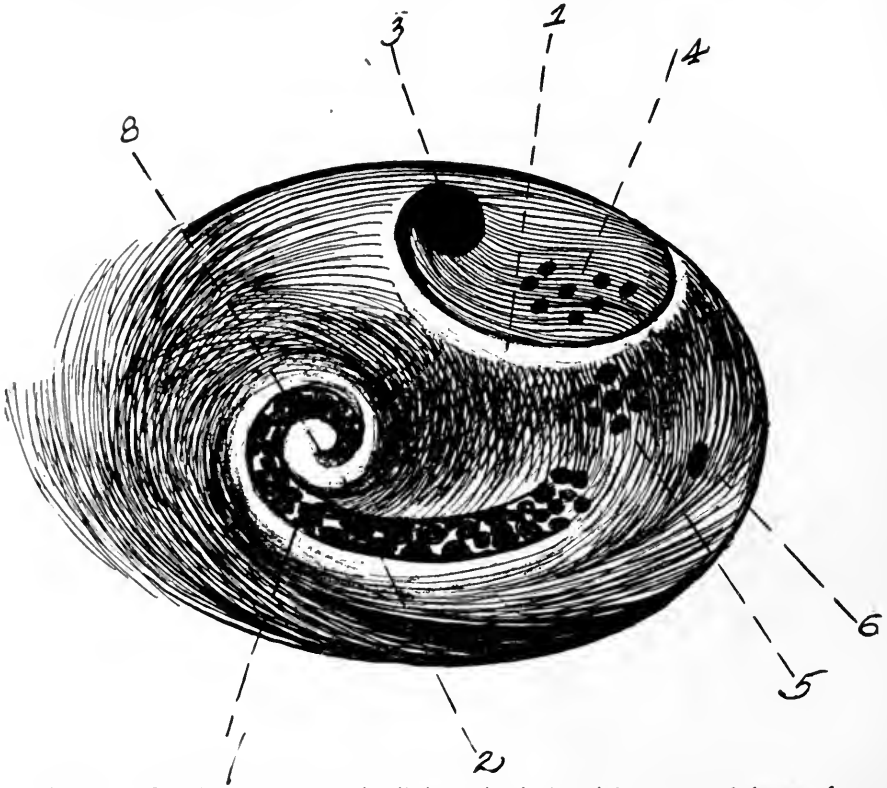


Figure 4.—Fundus meatus auditorii interni of the right temporal bone of a child—after Schwalbe.

1—Crista falciformis. 2—Spiral crista corresponding to the spiral turnings of the cochlea and defining the tractus spiralis foraminulentus. 3—Canalis N. facialis—opening for nervus facialis. 4—Area cribrosa superior for the passage of the branches of vestibular nerve which go to the macula sacculi. 5—Area cribrosa media for the passage of branches of vestibular nerve which go to macula utriculi. 6—Foramen singulare for the N. ampullaris posterior. 7—Tractus spiralis foraminulentus for the passage of the branches of the ramus cochlea. 8—Foramen centrale cochleæ.

between the canal and the surface of the bone directly posterior to the canal is quite acute (25°). At the depth of about 6 mm. from the posterior lip of the meatus in a plane almost at right angles to the di-

rection of the canal or, in other words, in the sagittal plane, the canal is partially obstructed by a perforated plate of bone. Through the perforations pass the VII (Facial) and the branches of the VIII (Acoustic) nerves. These perforations are illustrated in Fig. IV.

The **aqueductus vestibuli** viewed from below is a slit-like foramen on the posterior median surface of the petrous bone, about 1½ cm. external to and slightly below the level of the meatus. Immediately below it is a small, more or less rounded, depression for the accommodation of the saccus endolymphaticus.

The **aqueductus cochleae** is another slit-like foramen on the lower edge of the posterior median surface of the pyramid, immediately below the meatus acusticus internus; it serves for the exit of the ductus perilymphaticus from the scala tympani of the lower cochlear whorl previously mentioned.

MEMBRANOUS LABYRINTH.

The membranous labyrinth takes more or less the form of the osseous labyrinth with the exception of two sacs, the sacculus and utriculus, located within the cavity of the vestibulum (see Figs. V, VI and VII).

The membranous labyrinth is composed of four essential parts: (1) The Ductus Cochlearis, a membranous tube within the osseous cochlea, taking the same spiral turns and ending in a blind end at the apex (cæcum cupulare); (2) The Sacculus, a more or less pear shaped sac resting in the recessus sphericus of the vestibule; (3) The Utriculus, an irregularly ovoid shaped sac resting in the recessus ellipticus of the vestibule; (4) The three Semicircular Canals, corresponding and loosely adherent to the osseous canals. At one end of each of these semicircular canals as they enter the utriculus, is a dilatation known as the ampulla.

The membranous is much smaller than the osseous labyrinth; it contains a fluid known as the endolymph. Surrounding the membranous labyrinth is the perilymphatic space containing the perilymph.

The **ductus cochlearis** has two blind ends, the upper—cæcum cupulare, already mentioned—and a lower one projecting into the cavity of the vestibule and known as the cæcum vestibulare (see Figs. VI and VII). Very close to the latter is a small duct (ductus reuniens) leading from the ductus cochlearis to the sacculus. More about the ductus cochlearis need not be said in this paper since it does

not bear upon the subject of the physiology and pathology of the non-acoustic labyrinth.

The **sacculus** is a sac somewhat smaller in size than the utriculus and communicates with the ductus cochlearis through the ductus reuniens and indirectly with the utriculus through the ductus utriculosaccularis, which empties into the ductus endolymphaticus a short distance after the ductus endolymphaticus leaves the sacculus. Surrounding the sacculus is the perilymphatic space of the vestibule containing the perilymph.

The sacculus contains an important end organ for the perception of the static function, known as the macula acustica sacculi, which receives its nerve supply from the nervus saccularis, a branch of the nervus vestibuli.

The **utriculus** is located somewhat posterior and superior to the sacculus. It has six openings, one for the short ductus utriculo-saccularis, which empties into the ductus endolymphaticus, and five for the three semicircular canals; three of these receive the ampullar ends of the three canals, one the nonampullar end of the external semicircular canal, and one the crus commune, which represents the blending into one of the two smooth ends of the vertical canals.

The utriculus, like the sacculus, contains an end organ for the perception of the static function, known as the macula acustica utriculi and receives a corresponding branch (nervus utriculi) from the vestibular nerve.

The three membranous canals are much smaller, but correspond in form to the osseous canals; they are: (a) the ductus semicircularis lateralis, (b) superior, and (c) posterior.

The spindle shaped ampullæ of the three canals are known respectively as the ampulla membranacea lateralis, superior and posterior. Each ampulla contains an end organ for the perception of motion acceleration in curved lines; of this we shall speak further when we come to the physiology. These three end organs are located on a sort of spine running transversely to the canal within the ampulla on the more distant side (see Fig. IX).

The membranous canals contain endolymph which is capable of motion in two directions—from the canal to the utriculus, and vice versa. Surrounding the membranous canal is a space containing delicate fibrous threads, which hold the membranous canal more or less fixed to the more distant side of the osseous canals. This space is known as the spatium perilymphaticus and contains the perilymph.

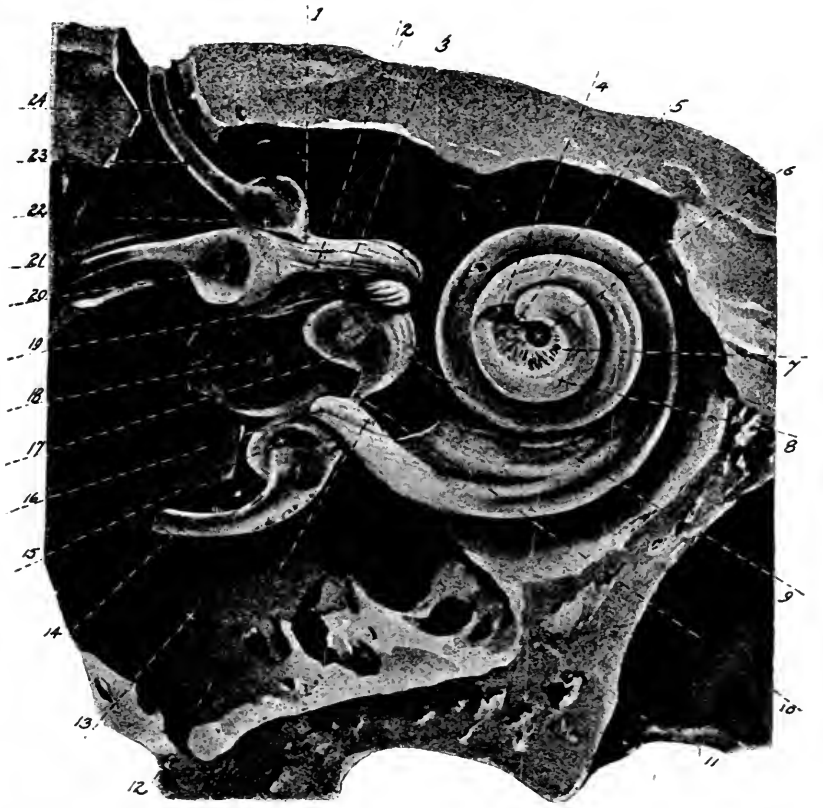


Figure 5.—Right membranous labyrinth of an adult partly laid free (from the lateral side) enlarged 5 diameters—after Spaltcholz.

1—N. ampullaris superior. 2—N. ampullaris lateralis. 3—N. utricularis. 4—Scala vestibuli. 5—Cæcum cupulare. 6—Helicotrema. 7—Hamulus ossis lacrimalis. 8—Ductus cochlearis. 9—Macula acustica sacculi with N. saccularis. 10—Ductus reuniens. 11—Canalis caroticus. 12—Ductus cochlearis. 13—Cut surface of the bone. 14—Ductus semicircularis posterior. 15—Ampulla membranacea posterior. 16—Cæcum vestibulare. 17—Sacculus. 18—Utriculus. 19—Macula acustica utriculi. 20—Ampulla membranacea lateralis. 21—Ductus semicircularis lateralis. 22—Ampulla membranacea superior. 23—Ductus semicircularis superior. 24—Canalis semicircularis superior.

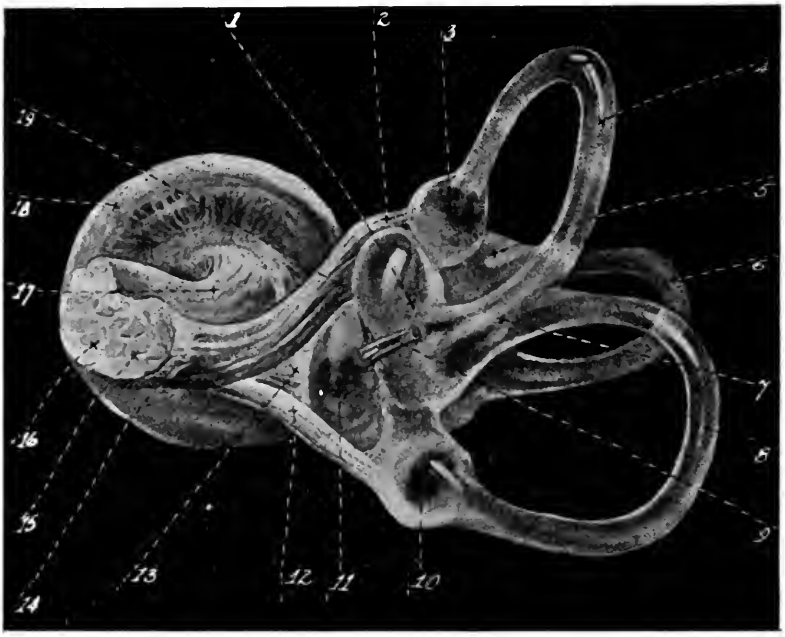


Figure 6.—Right membranous labyrinth of an adult laid bare, viewed from the median and posterior side, enlarged 5 diameters—after Spalteholz

1—Utriculus. 2—N. ampullaris superior. 3—Ampulla membranacea superior. 4—Ductus semicircularis superior. 5—Ampulla membranacea lateralis. 6—Ductus semicircularis lateralis. 7—Crus commune. 8—Ductus semicircularis posterior. 9—Ductus reuniens. 10—Ampulla membranacea posterior. 11—Sacculus. 12—N. ampullaris posterior. 13—N. saccularis. 14—Ganglion vestibulare. 15—N. vestibularis. 16—N. acusticus. 17—N. cochlearis. 18—Ductus cochlearis. 19—Superior branch of the vestibularis.

MICROSCOPIC ANATOMY.

In considering the microscopic anatomy of the nonacoustic labyrinth, the cochlea and its membranous contents are naturally excluded.

The nonacoustic labyrinth contains two types of nerve endings: (a) two of the one type located in the sacculus and utriculus are known as the macula acustica sacculi and the macula acustica utriculi; (b) three

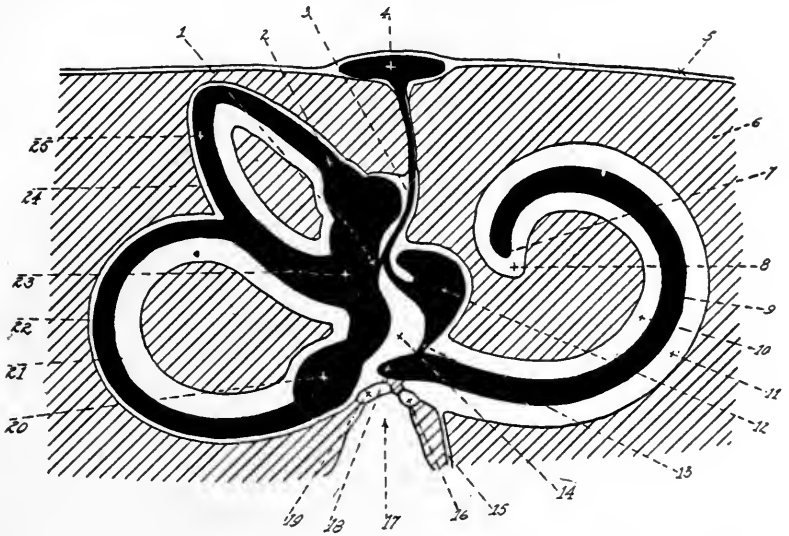


Figure 7.—Schematic representation of the osseous and membranous labyrinths, enlarged 5 diameters—after Spalteholz.

1—Ductus utriculosaccularis. 2—Ampulla membranacea superior. 3—Ductus endolymphaticus. 4—Saccus endolymphaticus. 5—Dura mater encephali. 6—Bone. 7—Cæcum cupulare. 8—Helicotrema. 9—Ductus cochlearis. 10—Scala vestibuli. 11—Scala tympani. 12—Sacculus. 13—Ductus reuniens. 14—Spatium perilymphaticum of the vestibule. 15—Ductus perilymphaticus. 16—Fenestra cochleae. 17—Cavum tympani. 18—Cæcum vestibulare. 19—Fenestra vestibuli. 20—Ampulla membranacea posterior. 21—Canalis semicircularis posterior. 22—Ductus semicircularis posterior. 23—Utriculus. 24—Canalis semicircularis superior. 25—Ductus semicircularis superior.

of another type located in the three ampullæ membranaceæ which are known as the cristæ ampullari.

It is these two maculæ and three cristæ which particularly interest us, and for this reason we shall describe their microscopic anatomy

somewhat in detail. A description of one macula will answer for both, as will also a description of one crista answer for the remaining two.

The **macula acustica** utriculi is located within the cavity of the utriculus. According to Retzius (das Gehororgan der Wirbeltiere, II., p. 333) the long axis is from above, in front and inward, to behind, below and outward, and the shorter diameter at right angles to this and somewhat horizontal. The long axis of the macula acustica sacculi, according to the same author, lies on the median wall and is al-

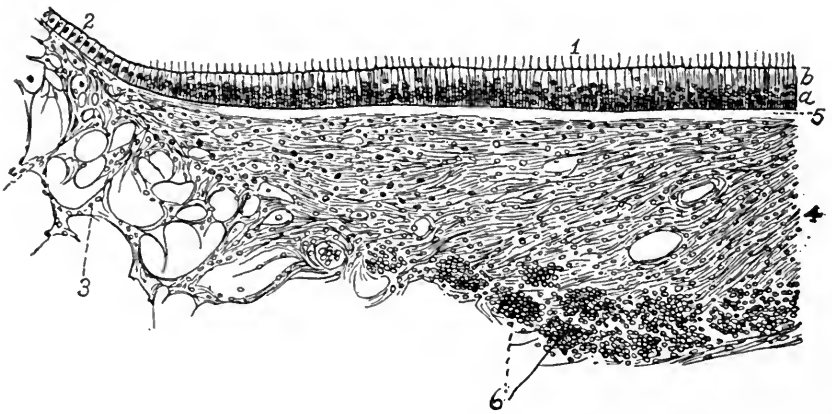


Figure 8.—Cut through the macula acustica recessus utriculi of the human, magnified 130 diameter.—after Schwalbe.

1—Neuro-epithelium of the macula acustica; a, nuclear zone; b, zone free of nuclei; upon the surface, ciliæ. 2—Cuboid cells at the border of the macula. 3—Perilymphatic net work of connective tissue trabeculæ. 4—The same but more compact. 5—The basement membrane. 6—Nerve bundles cut transversely.

most vertical, or more exactly from above and inward to below and outward, and its shorter diameter is at right angles to this direction.

The maculae receive the terminal nerve filaments of the nervi utriculi and sacculi, branches of the vestibular nerve. Each macula is composed of a layer of two distinct kind of cells—(a) neuro-epithelial cells; (b) supporting cells. The neuro-epithelial cells are the actual perceiving cells (special sense cells) and contain short hairs or cilia of uniform length extending into the cavity of the sacculus and utriculus. The supporting cells are the alternate ones between the perceiving cells; they contain no cilia and have no known function but are supposed to support, and the writer would add to insulate, the perceiving cells.

On the surface of the cells or the cilia of the cell rest somewhat loosely the socalled otoliths, small crystalline bodies which are held more or less together and to the surface by a mucilaginous substance which prevents them from becoming separated or detached from the surface through change of position of the head, when for instance the surface of the macula is above and the otoliths below. The macula and otoliths are nothing more than a more complex and higher developed aural vesical or otocyst with the otoliths found in the craw fishes

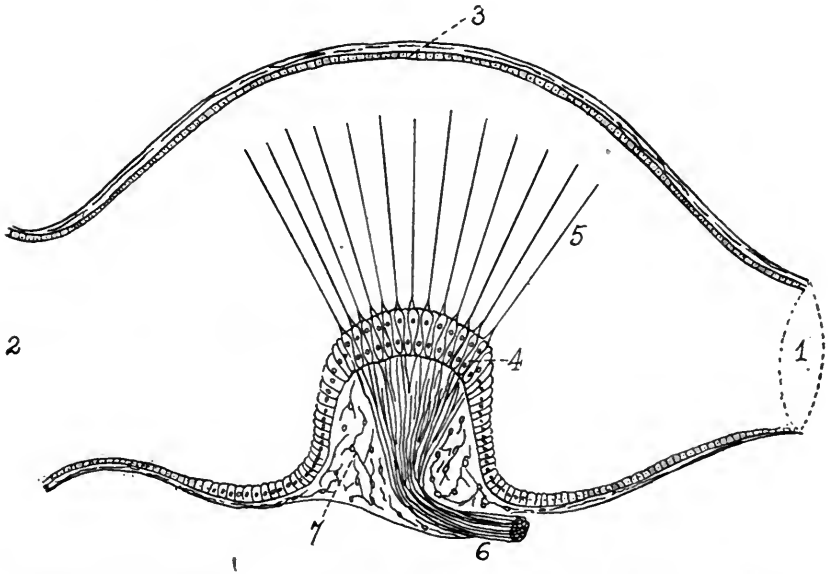


Figure 9.—Longitudinal cut through the ampulla—after Hensen (schematic). 1—Semicircular canal communication. 2—Utriculus communication. 3—The epithelia of the ampullar roof. 4—Neuro-epithelia of the crista ampullaris or acustica. 5—The long cilia of the neuro-epithelia. 6—N. ampullaris. 7—Connective tissue of the crista.

which Kreidl has demonstrated to be the static organ by the iron filings and magnet experiment.

Crista ampullaris. There are three cristæ; one for each membranous ampulla. They are located on the far side of the ampullæ on the summit of the prominences which project into and narrow the lumen of the ampullæ. These prominences are really ridges which appear triangular-shaped when viewed from the side and more or less crescent shaped when viewed lengthwise of the canal; in other words,

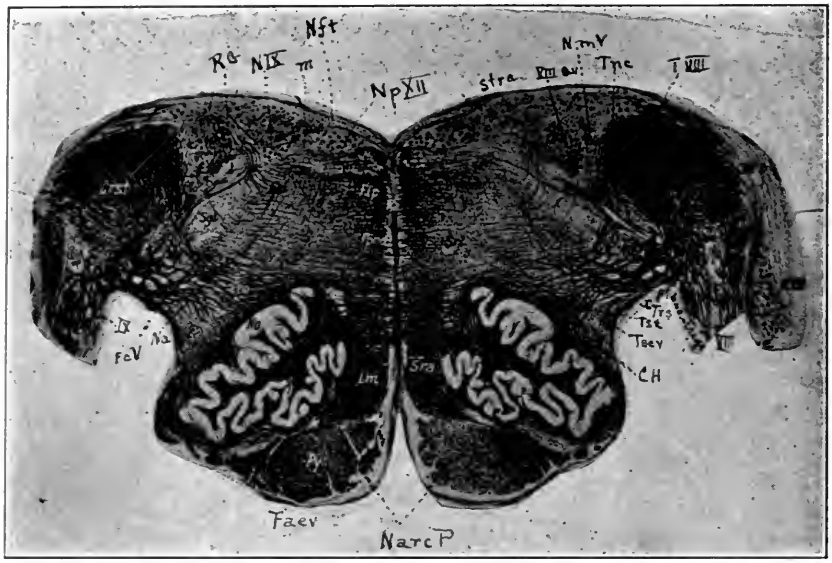


Figure 10.—Transverse cut through the medula oblongata showing the origin of the nervus acusticus, the Deiter's and the triangular nuclei. (From the Mikroskopisch-topographischer Atlas des menschlichen Zentralnervensystems—after Marburg.)

Stra Striæ acusticæ. VIII av Pars vestibularis radicis spinalis acustici. Nmv Nucleus vestibularis magnocellularis. Tnc Tractus nucleo-cerebellaris. TVIII Tuberculum acusticum. VIIIc Ramus cochlearis asustici. NVIIIac Nucleus acustici acessorius. VIII Nervus acusticus. Crst Corpus restiforme. Flp Fasciculus longitudinalis dorsalis. RG Area glossopharyngei. NIX Nucleus glossopharyngei dorsalis. m Fasciculus longitudinalis dorsalis. Nft Nucleus eminentiæ teretis. Np XII Nucleus praepositus hypoglossi. pt Fibræ pretrigeminales. Trs Tractus rubrospinalis. Tst Tractus spinotectalis et thalamicus. Tscv Tractus spinocerebellaris ventralis. cH Fasciculus tegmeti Centralis. NarcP Nucleus arcuatus pyramidum. Py Pyramis. faev Fibræ arcuatæ externæ ventrales. Na Nucleus ambiguus. F c V Fasciculi comitantes trigemini. IX Nervus glossopharyngeus. Va Radix spinalis trigemini. Sgl Substantia gelatinosa trigemini. fai Fibræ arcuatæ internæ. Fco Fibræ cerebello olivares. No Nucleus olivaris inferior. Oae Paroliva dorsalis. Lm Lemniscus dorsalis. Sra Substantia reticularis alba. Srl Substantia reticularis lateralis. Nci Nucleus centralis inferior. Ra Raphe. Fprd Fasciculi predorsalis. rt Fibræ retrotrigeminales. it Fibræ arciformes intertrigeminales.

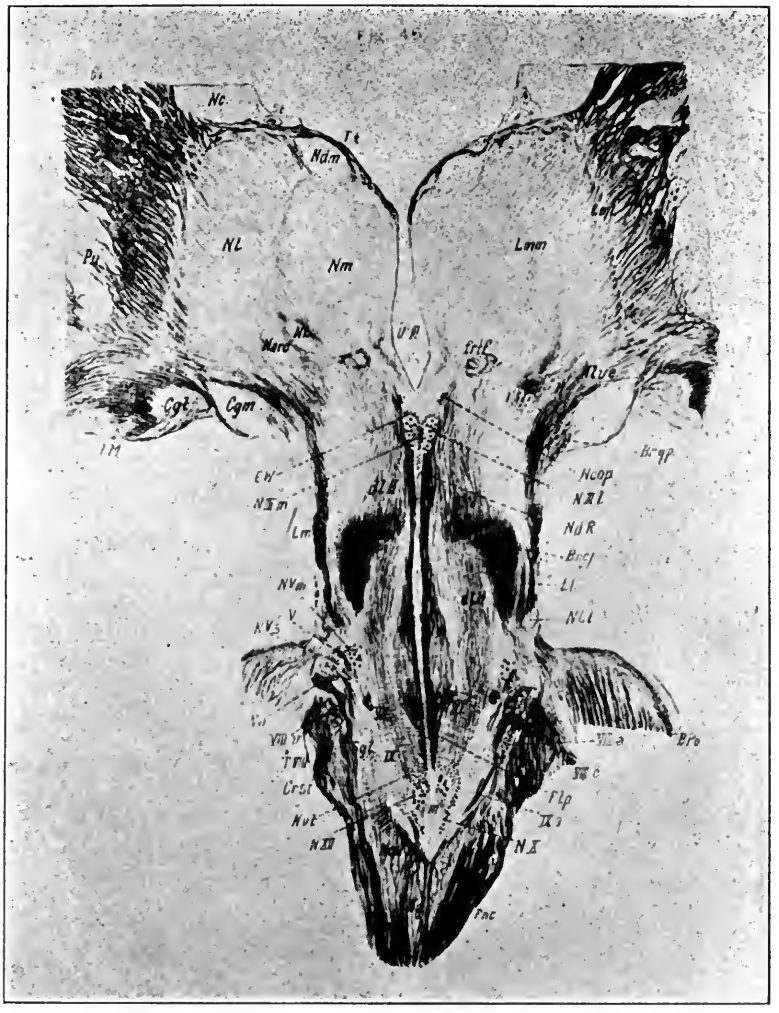


Fig. 11. Longitudinal cut through the medulla oblongata corpora quadrigemina and thalamus opticus, showing Deiter's Nucleus, the eye muscle. nuclei and the posterior longitudinal bundle. (From the Mikroskopisch-topographischer Atlas des menschlichen Zentralnervensystems—after Marburg).

E W Nucleus parvicellularis oculomotorii. NIIhn Nucleus oculomotorii medialis. NIIII Nucleus oculomotorii lateralis. VIIIv Ramus vestibularis acustici. T VIII Tuberculum acusticum. Nvt Nucleus abducens. Flp Fasciculus longitudinalis dorsalis. VIII e Ramus cochlearis acustici. VIII a Radix spinalis acustici. Ci Capsula interna. Nc Nucleus caudatus. St. Stria cornea. Ndm Nucleus dorsalis magnus thalami. Tt Taenia thalami. Pu Putamen. Nl Nucleus lateralis thalami. Nm Nucleus medialis thalami. Narc Nucleus arcuatus thalami. Nl Corpus Luyisii. Cgm Corpus geniculatum mediale. Cgl Corpus geniculatum laterale. IM Meditullium lateralle. VIII Ventriculus tertius. Lmm Lamina medullaris lateralis. frtf Fasciculus retroflexus. Ntg Nucleus riber tegmenti. H. Fasciculus triangularis Helweg. Nlve Nucleus lateralis externus thalami. Brqp Brachium corporis quadrigemini posterioris. Ncop Nucleus commissuræ posterioris. Ndr. Nucleus dorsalis raphes. Brcj Brachium conjunctivum. Ll Lemniscus lateralis. NLl Nucleus lemnisci lateralis. dl H Fasciculus tegmenti dorsolateralis. Lm Lemniscus medialis. N Vm Nucleus motorius trigemini. V Nervus trigemini. N Vs Nucleus sensibilis trigemini. Va Radix spinalis trigemini. Crst Corpus restiforme. NXII Nucleus hypoglossi. Ncu Nucleus funiculi cuneati. Ng Nucleus funiculi gracilis. Fnc Fasciculus cuneatus. m Fasciculus longitudinalis dorsalis. Sgl Substantia gelatinosa trigemini. VLL Nervus facialis. LX Nervus glossopharyngeus. NX Nucleus vagi dorsalis. LXa Radix spinalis glossopharyngei. BPo Brachium pontis.

the long axis of the ridge is at right angles to the long axis of the canal. The ridge is built up of a fan-like separation of the ampullar nerve filaments plus connective and vascular tissues. The nerve filaments terminate in the special sense end-organ (crista ampullaris).

The cristæ, like the maculæ, are composed of ciliated neuro-epithelial and supporting cells. The ciliated cells are the real special sense or perceiving cells. These differ from those of the maculæ in that they carry much longer cilia.

The supporting cells of the cristæ no doubt perform a similar function to the supporting cells of the maculæ.

The cilia are held more or less together, or so to speak matted together, by a mucilaginous substance in such manner that their distal tips come close together, much like a paint brush after dipping in oil, and looks not unlike it. On the summit of the cilia is an accumulation of the mucilaginous or gelatinous substance which is known as the cupula terminalis. The cilia are more or less flexible and capable of inclination in two directions—toward and away from the utriculus in the long axis of the canals. Because of the matting together of the cilia they all move in one or the other direction as a single mass.

All of these five end organs receive their nerve supply from branches of the vestibular nerve, the course of which according to Obersteiner and Marburg is as follows:—the peripheral fibers are interrupted and terminate in the bipolar ganglion cells (ganglion vestibulare or Scarpa's ganglion). From this ganglion originate central fibers which unite into a thick trunk and form the median acusticus root (vestibular). This root of VIII N. enters the medulla oblongata at the level of the olive, just medianward of the nucleus accessorius. The fibers then pass in a median dorsal direction between the corpus restiforme and the spinal trigeminus root to the nucleus vestibularis triangularis and the nucleus vestibularis magnocellularis. The latter nucleus comprising Deiter's and Bechterew's nuclei. (Figs. 10 and 11.)

The nucleus triangularis and the nucleus magnocellularis lie on the floor of the fourth ventricle; the nucleus triangularis somewhat more medianward and nearer the floor of the ventricle than the nucleus magnocellularis.

From the triangularis nucleus go ascending and descending fibers.

From the nucleus magnocellularis go both crossed and uncrossed, ascending and descending fibers. The ascending fibers form the posterior longitudinal bundle—fasiculus longitudinalis dorsalis of Schulz,

and are in direct communication with the nuclei of the VI, IV and III nerves. It is this part of the so-called Deiter's system which has to do with vestibular reflex nystagmus.

Descending fibers from the nucleus magnocellularis go to the motor cells of the anterior horns of the spinal cord, and they play a part in the co-ordination of the trunk and leg muscles.

Furthermore from the nuclei vestibuli magnocellularis et triangularis go fibers by the tractus nucleocerebellaris to the cerebellum. These fibers terminate in the nuclei tecti of the same and opposite side, and according to some authors to the cortex of the cerebellum.

RELATIVE POSITIONS OF THE SEMICIRCULAR CANALS.

The writer considers the relative position of the semicircular canals to the skull and to each other a subject of some importance, since quite a number of men (beginners) with whom he has spoken concerning the labyrinth have had the erroneous impression that the lateral or horizontal canals lie in the horizontal, the superior canal in the frontal and the posterior in the sagittal planes of the skull, and these same men are prone to associate horizontal nystagmus with the horizontal canals, rotatory nystagmus with the superior canals and vertical nystagmus with the posterior canals. For this and other reasons he feels it necessary to describe and illustrate the realtive positions of the canals.

In the erect position of the head the external canals lie practically in the horizontal plane; furthermore, the planes of the two sides are one and the same. If the lateral canals of the two sides were united they would make a complete circle, the two smooth ends meeting and blending posteriorly and the two ampullæ coming together anteriorly.

The two superior canals lie in vertical planes, but unlike the external canals, they are neither in the same nor parallel planes; but on the contrary, the planes of the canals of the two sides are at right angles to each other, the planes meeting in the median line posteriorly at the point P, at the posterior margin of the foramen magnum. Again, when the canals of the two sides are united we do not have the arrangement (blending of the smooth ends and meeting of the ampullar ends) as in the case of the external canals.

The two posterior canals lie in vertical planes at right angles to each other, meeting at the point A—Fig. 12—in the median line in the region of the sella tursica. Again, if these canals were united we would not have the same arrangements as in the case of the external canals.

When considering the two superior or two posterior canals together there is a lack of congruity or association which we find existing in the case of the two external canals. On the other hand, if we consider the superior canal of one side with the posterior canal of the opposite

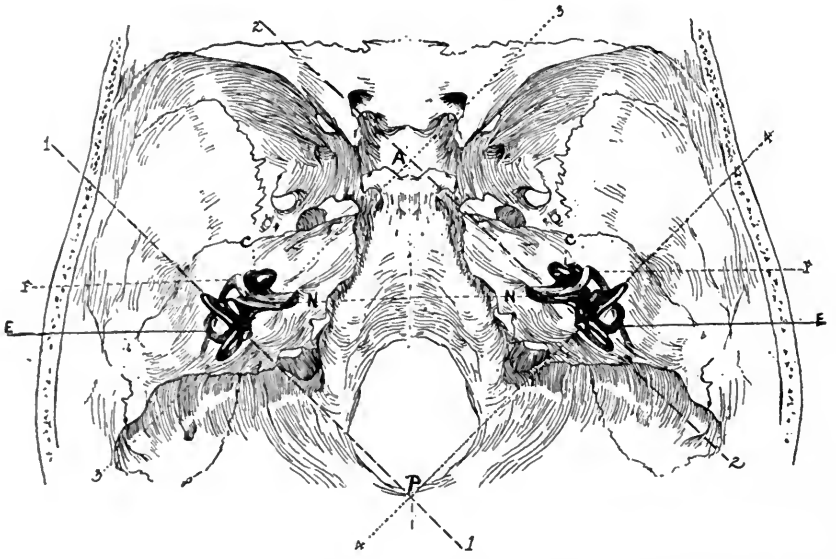


Figure 12.—Half schematic representation of the two labyrinths showing the relative positions of the semicircular canals to the skull and to each other.

N—7th and 8th nerves as they enter the internal auditory foramen. C—Cochlea. F—Geniculate ganglion of the facial nerve. E—External semicircular canal. The dashed line 1-1 illustrates the vertical plane of the left superior semicircular canal. The dashed line 2-2, the vertical plane of the right posterior semicircular canal. The dotted line 3-3 represents the vertical plane of the left posterior semicircular canal. The dotted line 4-4 represents the vertical plane of the right superior semicircular canal. A—The point of meeting of the vertical planes of the posterior semicircular canals of the two sides. P—The point of meeting of the vertical planes of the superior semicircular canals of the two sides.

side, their association as found in the external canals is established. In other words, they lie in parallel planes, and if the canals are brought together they would form a circle with the smooth ends coming together and blending, while the two ampullæ would meet at a point 180° from the smooth ends. Thus we see that the six canals of the two sides are correllated and designed after a definite plan.

PHYSIOLOGY AND PATHOLOGY OF THE NONACOUSTIC OR, SO-CALLED, STATIC LABYRINTH.

NYSTAGMUS.

BEFORE considering that special form of nystagmus which may be produced physiologically by irritation of the nonacoustic labyrinth or pathologically by irritative or destructive changes in the same, let us briefly consider the subject of nystagmus in general.

Nystagmus is an involuntary to and fro movement of the eyes, subject in some instances to more or less voluntary inhibition.

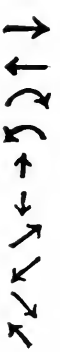
According to the plane in which the movements take place we have horizontal, rotatory, vertical or oblique nystagmus; or any combination of these. According to the comparative rapidity of the two movements (to and fro) we have the oscillatory and rhythmic forms of nystagmus.

In the case of oscillatory—also known as undulatory—nystagmus the two component eye movements or excursions are performed with *equal* rapidity, just as in the case of the pendulum of a clock, the oscillation to one side is performed with precisely the same rapidity as the oscillation to the opposite side. For this reason we cannot speak of oscillatory nystagmus taking place in any definite direction. We may speak of a horizontal oscillatory nystagmus, but it would be nonsense to speak of a horizontal oscillatory nystagmus to the right or to the left.

In the case of rhythmic nystagmus the two component eye movements are performed with *unequal* rapidity; in other words, the eye movements are jerky or spasmodic in one direction and slow in the opposite; for this reason we may consistently speak of horizontal rhythmic nystagmus to the right or left.

Authors have agreed to specify the direction of rhythmic nystagmus according to the direction of the rapid movement; thus horizontal nystagmus to the right is understood to be rhythmic and not oscillatory, from the fact that a definite direction is specified; the plane of the movements is horizontal and the direction of the quicker movements is to the right.

For the sake of convenience the plane and direction of rhythmic nystagmus may be indicated by appropriate signs, as follows:



L. Horizontal nystagmus to the patient's left.
R. Horizontal nystagmus to the patient's right.
L. Rotatory nystagmus to the patient's left.
R. Rotatory nystagmus to the patient's right.
U. Vertical nystagmus upward.
D. Vertical nystagmus downward.
U. L. Oblique nystagmus upward and to the patient's left.
D. R. Oblique nystagmus downward and to the patient's right.
D. L. Oblique nystagmus downward and to the patient's left.
U. R. Oblique nystagmus upward and to the patient's right.

The directions indicated by the arrows above correspond to the directions of the eye movements as seen by the observer looking directly at the patient, from in front. These signs are the author's, modified after Barany, and have been adopted and used by Alexander and his staff in the ear department of the Polyclinic in Vienna.

It is well at this time to differentiate still further the oscillatory and rhythmic forms of nystagmus, including a review of the points already mentioned.

OSCILLATORY NYSTAGMUS.	RHYTHMIC NYSTAGMUS.
1—May take place in any plane.	1—May likewise take place in any plane.
2—The two opposite movements occur with *equal* rapidity.	2—The two opposite movements occur with *unequal* rapidity.
3—We *cannot* speak of a definite direction of this nystagmus.	3—We *may* speak of a definite direction, corresponding to the direction of the rapid movement.
4—*Cannot* be produced experimentally.	4—*May* be produced experimentally by active or passive rotations, by caloric, mechanical or galvanic irritation of the ear labyrinth (vestibular); or by the looking out of a moving train, looking at a waterfall, etc. (optical.)

5—*Is not* accompanied with vertigo.	5—*Is generally* accompanied with vertigo, at least when the nystagmus first occurs.
6—*Is always* of pathologic significance.	6—*Is not always* of pathologic significance, since it may be produced physiologically by attempts at voluntary fixation of the gaze in any extreme direction. Besides, most optical forms (see above) are generally physiologic.
7—The intensity *can be* voluntarily inhibited, and in some cases even to the extent of bringing about temporary cessation. The inhibition of the nystagmus is in direct proportion to the inhibition of the attempt at central visual fixation.	7—The intensity of the nystagmus *can be* inhibited *only* by the patient looking in the opposite direction to his nystagmus. A pronounced nystagmus to the right when looking to the right *cannot* be inhibited so long as the patient continues to look in that direction.
8—Oscillatory nystagmus is practically a *permanent* pathologic condition, and tends in some cases to *increase.*	8—Rhythmic nystagmus is generally a *transient* condition, and tends rather to *diminish* and may even cease in some cases.
9—Oscillatory nystagmus, in some cases, has been observed to take on a somewhat rhythmic character during attempts at fixation in extreme lateral positions.	9—On the contrary, rhythmic nystagmus *cannot* be made to take on an oscillatory character.

Nystagmus is almost always bilateral. In exceptional cases we find it unilateral; in other exceptional cases, tho the nystagmus is bilateral and synchronous, the length of the nystagmic excursions are unequal in the two eyes. In this connection the writer will cite three cases illustrating these variations.

CASE I.—A woman about 50 years of age, with both labyrinths normal, was examined upon the revolving stool. After ten complete turns to the left with the head in the upright position, instead of manifesting a bilateral horizontal after-nystagmus to the right—as the vast majority of cases do—she manifested a horizontal after-nystagmus to the right *of the right eye only*, the left eye remaining stationary. The same results were obtained when turned in the opposite direction, *i. e.*, after-nystagmus to the left of the left eye only, the right eye remaining stationary.

With the caloric test the same character of reactions was obtained, namely, unilateral nystagmus and always of the eye of the side to which the nystagmus manifested itself.

CASE II.—A woman, aged 40, of low intelligence, after the turning test (as above described) manifested instead of nystagmus a deviation of one eye. For instance: Instead of manifesting a bilateral nystagmus to the right by the various tests which produce nystagmus to the right, she manifested a *deviation of the right eye to the left*, in other words, a convergent strabismus of the type corresponding to paralysis of the right external rectus. The deviation endured for a length of time corresponding to a nystagmus reaction in the average normal individual.

CASE III.—A young man 21 years old, with diffuse corneal opacity in the right eye and slight microphthalmos of the same eye, manifests a *pronounced* spontaneous rhythmic nystagmus of the right eye to the left; while the left normal eye manifests a *barely perceptible* nystagmus synchronous with that of the right eye.

ETIOLOGY OF OSCILLATORY NYSTAGMUS.

Oscillatory nystagmus is usually, if not always, due to congenital or early acquired visual defects; especially central visual defects. We therefore find it prone to occur in cases of centrally located opacities of the ocular media (central corneal macula, etc.) and in cases of fundus disease where the lesion involves the region of the macula lutea, more especially when these conditions have occurred before or about the age at which a child learns to fix the eyes for central vision. In other words, the more central the visual defect and the earlier in life it occurs, the more likely the child is to develop oscillatory nystagmus. While the above conditions favor most the development of oscillatory nystagmus, we find cases developing later in life. We also find rare cases of congenital oscillatory nystagmus occurring in individuals where every method of examination fails to reveal any pathologic condition of the eyes; but on the other hand, they show at least some defect of central vision. The cause in these latter cases is unknown; however, it is possible that at some future time they

may be found to result from some congenital hemorrhage or other character of lesion in the tracts or centers corresponding to central macular vision.

While oscillatory nystagmus results from congenital or early acquired visual defects, not every case of congenital or early acquired visual defect produces the oscillatory form of nystagmus, for we find cases where the resulting nystagmus is rhythmic in character. The writer has examined such cases for the purpose of determining the presence of other factors that might have caused the nystagmus to be rhythmic instead of oscillatory in character, but with negative results.

Concerning the exact causes in all cases of oscillatory nystagmus we have yet much to learn. Fortunately for the otologist, the causes of rhythmic nystagmus have been more definitely ascertained.

ETIOLOGY OF RHYTHMIC NYSTAGMUS.

Rhythmic nystagmus may be produced physiologically, pathologically or experimentally.

Physiologically, as already pointed out, by looking out of a moving car window at passing objects (horizontal nystagmus to the left when looking out of the right side of the car which is moving forward); by looking out of elevator car (vertical nystagmus upward when car is going up, and vertical nystagmus downward when car is going down). Again, these same nystagmi may be produced physiologically by one remaining stationary and observing moving objects—waterfalls (vertical upward), moving carousal (horizontal to the right when the carousal is moving to the left past our field of vision, etc.). All of these represent physiological optical nystagmus.

Rhythmic nystagmus may be produced physiologically also by active or passive rotation of the head in any plane. Barany has found that a single rotation will produce nystagmus in the plane of rotation and in the direction of rotation during the turning, *even with the eyes closed.* With the eyes open the nystagmus produced by rotation represents a combined optical and vestibular nystagmus; with the eyes closed it represents purely vestibular nystagmus.

The nystagmus manifested by attempts at fixation of the eyes in extreme positions may in a sense be termed physiologic. For instance all normal individuals when looking to the extreme right or left manifest rhythmic nystagmus to that particular side. This nystagmus is due to the extra strain put upon the abductors of one eye and the adductors of the other eye, and compares with the clonic contractions of other

body muscles when any extra strain is put upon them. This nystagmus is dependent upon two contrary working factors: (1) the strong voluntary cortical impulse to the muscles, and (2) the inherent weakness of the muscles to maintain the excessive muscular contraction. The contractions are intermittent, with the result that the individual manifests nystagmus. This form of nystagmus varies in different individuals; for instance, those with weak eye muscles and with strong power for voluntary cortical innervation will manifest a more marked nystagmus than those with stronger muscles and weaker power of innervation. It is characteristic for nystagmus of this type to manifest itself with equal intensity to both sides.

The knowledge of all these facts is important and will save much confusion when we come to consider the observations of vestibular nystagmus.

Rhythmic nystagmus may be produced pathologically by unilateral, irritative or destructive conditions of the labyrinth, the vestibular nerve, its nucleus in the medulla, the supranuclear tracts (posterior longitudinal bundle and tractus nucleo-cerebellaris) or of the cerebellum itself, which involve the sensory or centripetal impulses. Again, rhythmic nystagmus may be produced pathologically by irritative or destructive lesions—of the supranuclear motor tract (including the posterior longitudinal bundle, which is really more motor than sensory), the cortico-nuclear, equivalent to the pyramidal tract, the motor nuclei, the motor nerves or the eye muscles themselves—which involve the motor or centrifugal impulses.

Besides the location, the nature of the lesions capable of producing nystagmus are numerous.

These two conditions (location and character of the lesion) taken together multiply the number of possible pathologic processes capable of producing rhythmic nystagmus, some of which may be cited, *e. g.:* any form of lesion of the labyrinth; hyperemia, inflammation; fistula; traumatism, direct and indirect, involving the labyrinth capsule and including fracture at the base of the skull passing thru the pyramid; hemorrhages into the labyrinth from chlorosis, pernicious anemia, from changes in atmospheric pressure in caisson workers, balloonists, etc.; ischemia from embolism or partial obliteration of the arteria auditivæ internæ in arteriosclerosis and endarteritis; toxemias from quinine, salicylates, etc.; granulomatous infiltration of syphilis, tuberculosis and leukemia.

Diseases of the vestibular nerve: neuritis; tumor; granulomatous

infiltration; basal meningitis; increased intracranial pressure in case of acute hydrocephalus, cerebral tumors and abscess.

Diseases of the medulla oblongata involving the vestibular nuclei (triangularis and magnocellularis), including bulbar and pseudobulbar palsy, tumors, syphilis, tuberculosis, hemorrhage, multiple sclerosis; toxic substances circulating in the blood stream (alcohol, nicotine, excess of carbon dioxid, etc.).

Diseases of the cerebellum: tumors, abscess, areas of multiple sclerosis, etc.

Diseases of the posterior longitudinal bundle: tumors, gummatous or tuberculous infiltrations on the floor of the fourth ventricle (above the striæ acousticæ) or the aqueductus silvii, multiple sclerotic plaques, etc.

Diseases of the eye muscle nuclei: tumors, hemorrhages, degenerations, abscess, sclerosis, emboli, arteriosclerosis, etc.

Diseases of the motor nerves of the eye muscles: neuritis from various causes (refrigeratory, postinfectious, toxic, luetic, etc.), tumors and granulomatous infiltrations, orbital fractures and intraorbital disease which might impair the innervation or action of a particular muscle or set of muscles.

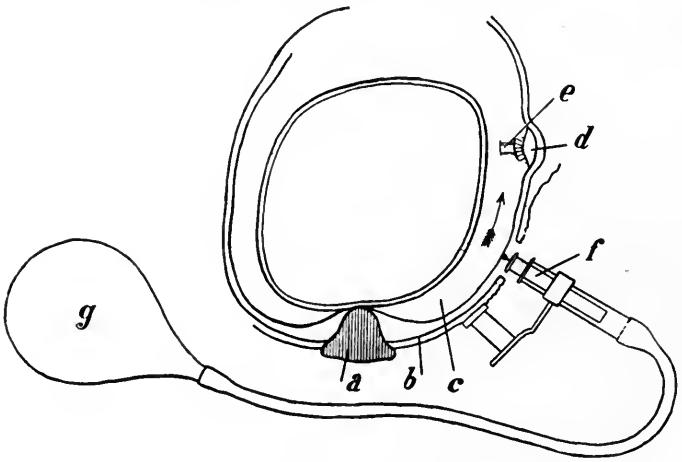
The writer has merely mentioned some of the pathologic causes; there are perhaps as many more, including the yet undiscovered, that may produce rhythmic nystagmus.

It might be said in this conjunction that the nystagmus of cerebellar disease is not a real ataxia nor is it homologous to the ataxia manifested in the extremities or trunk, as might be believed, but is a true rhythmic nystagmus. Marburg and others, including the writer, believe that the nystagmus of cerebellar disease is actually vestibular in type and originates from influences acting directly upon Deiter's nucleus, which lies in the immediate neighborhood of the lateral cerebellar lobe.

Experimentally optical nystagmus can be produced by the rotations of objects in various planes before the open eyes (experiments similar to those made by Mach) or by passive rotations of the individual in various planes about stationary surroundings. In this latter instance the resulting rotational nystagmus is produced by a combination of two irritations: (1) the passage of moving objects before the open eyes (optical), and (2) motion of the endolymph in the semicircular canals (vestibular). The elimination of the vestibular element and the production of pure optical rotational nystagmus after

the above method is possible only in those cases of deaf mutes which have complete destruction of both labyrinths or nerves. The elimination of the optical element and the production of pure vestibular rotational nystagmus is possible by the simple closure of the eyes during the rotations. In fact, this was one of the earlier methods of examination for rotational vestibular nystagmus, the determination of which was accomplished, during the turning, by feeling of the eye movements with the fingers thru the closed lids.

Experimentally, vestibular nystagmus can be produced in man by active and passive turnings, by caloric irritation of the labyrinth and by galvanic irritation of the labyrinth and nerve, or by mechanical irritation as practiced first by Ewald and later by Dreyfuss.



Sketch After Barany.

Ewald, 1888 (Zur. Physiologie der Bgg.—*Tageblatt de* 61 *Versammlung Deutscher Naturforscher u. Aerzte in Koeln,* von 18-23, Sept., 1888, s. 74-76), described for the first time his experiments upon pigeons made with his celebrated pneumatic hammer. This experiment was unique in the history of the subject and a description of his hammer and methods of applying it is well worth citing.

Ewald first exposed and prepared the osseous external semicircular canal of a pigeon. He then drilled a small opening thro it several mm. posterior to the ampulla, at the same time avoiding injury to the membranous canal. Through this opening he inserted a filling material similar to that used by dentists, which when it becomes set acted as a plug interrupting completely the lumen of the membranous canal at this point (a).

Next he made a second small opening after the manner of the first midway between the first opening and the ampulla. At the second opening he adjusted an apparatus known as a pneumatic hammer. This hammer consists of a slender glass cylinder open at both ends, inside of which glides a piston with a small button on its distal end, which comes in contact with the membranous canal and serves to give it the hammer effect. Over the other end of the glass cylinder is fitted a rubber tube of convenient length and on the end of this again is fitted the rubber bag (g). Compression of the bag held in the hand, drives the piston inward against the membranous canal. Relaxation of pressure on the bag produces aspiration, causing the piston to be withdrawn to its primary position. When the piston is driven inward by compression the displaced endolymph, since it cannot flow posteriorly on account of the plug (a), must flow anteriorly toward the ampulla and utriculus indicated by the arrow. On the other hand, by aspiration the endolymph must flow in the opposite direction. Ewald, after this manner of examination, found that compression, equivalent to movement of endolymph from the nonampullar toward the ampullar end, caused a slow movement of the eyes and head to the opposite side in the plane of the canal (horizontal); and that aspiration, equivalent to a movement of the endolymph from the ampullar toward the nonampullar end, caused a slow movement of the eyes and head to the same side.

From this, Ewald was able to conclude that definite movements of the endolymph in the horizontal canal were responsible for definite reflex movements of the head and eyes. Ewald after the same manner demonstrated the characteristic movements of the head and eyes corresponding to definite endolymph movements in the remaining canals.

The physiology of vestibular nystagmus produced by turning and the caloric irritation of the labyrinth differs from that produced by galvanism. Before discussing the physiology of nystagmus produced by these separate irritations, let us consider the physiology of vestibular nystagmus in general.

PHYSIOLOGY OF THE TWO MOVEMENTS OF NYSTAGMUS.

It is a well known and established fact that irritation of the semicircular canals of one side will produce nystagmus to that side and that the inhibition of impulses from the semicircular canals of one side will produce nystagmus to the opposite side. Let it be understood early that nystagmus produced by either irritation or inhibition is not the actual vestibular reflex, but merely the evidence of the reflex. The

actual reflex, from irritation, for instance, of the right semicircular canals, produces a deviation of the eyes to the left, while the quick, jerky movements of the eyes to the right (*Einstellung* of the German authors) is the result of a voluntary effort on the part of the individual to restore macular fixation of external objects which had been temporarily lost during the reflex deviation of the eyes.

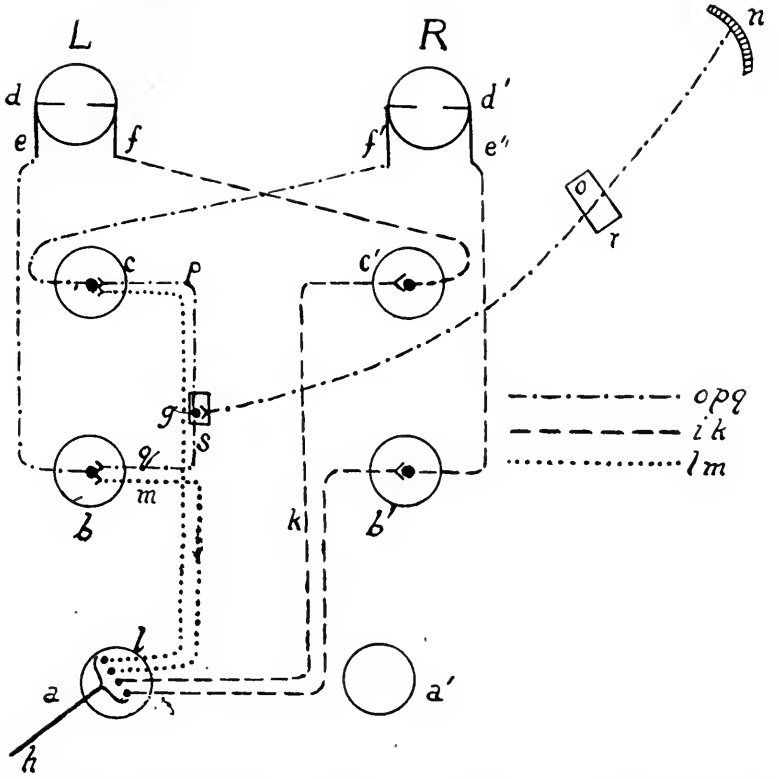
To better show the physiology of the two movements of nystagmus the writer will make use of an illustration from Barany's work upon the Phys. and Path. des Bgg. Appar. beim Menschen, page 53. See Fig. 14.

There is an abundance of evidence to show that the conjugate deviation of the eyes (slow movement of the nystagmus) is the actual vestibular reflex, and that the rapid movements in the opposite direction are purely voluntary, produced by cortical innervation.

In the making of various animal experiments by sectioning of the canals, by sectioning of the acusticovestibular nerve, by mechanical (Ewald's pneumatic hammer experiment) or by galvanic irritations of the semicircular canals, it has been found that some animals show only the reflex deviation (correspondng to the slow movements of the nystagmus) to the opposite or nonirritated side, while others will show rhythmic nystagmus to the same or irritated side. Again, the same animal will show by the same experiment at one time deviation and another time nystagmus. Frequently the nystagmus will be more pronounced immediately after the irritation or destruction, whereas later, as the animal tires, it will show only the deviation. Furthermore, it will be found that the lower the animal the weaker is the volition and the more likely it is to show only the reflex deviation; while the higher the animal, the stronger is the volition and the more likely it is to show nystagmus.

Again, if one experiments with a sufficient number of human subjects he will observe quite a difference in different individuals. It will be observed that the more intelligent people will show the more pronounced nystagmus; while occasionally very young children or very stupid people of low intelligence will manifest deviation only. When making time experiments of after-turning nystagmus, in those cases manifesting deviation only it is important to time the duration of the deviation just as one would in another case time the duration of nystagmus.

Fig. 14 illustrating horizontal nystagmus to the left produced by irritation of the left semicircular canal apparatus.



a, a¹ Nucleus Deiters, sinis, et dext. b, b¹ Nucleus nerv. abducens, sinist. et dext. c, c¹ Nucleus nerv. oculomotor, sinist. et dext. d, d¹ Oculus, sinist. et dext, e, e¹ Musc. rect. extern. sinist. et dext, f, f¹ Musc. rect. intern. sinist. et dext. g, Shaltzelle (Monakow) = centre for the movement of the eyes to the left. h, Nervus vest. sinist. i, Vestibular tract (— — — —) crossing to the contralateral abducens nucleus. k, Vestibular tract (— — — —) crossing to the contralateral oculomotorius nucleus. l, Vestibular inhibition tract (.....) to the homolateral oculomotorius nucleus. m, Vestibular inhibition tract (.....) to the homolateral abducens nucleus. n, Gyrus angularis dextra (cortex), center for the voluntary movement of the eyes to the left. o, Cortical tract (—.—.—.—.) to subcortical center (Monakow's) for the voluntary movements of the eyes to the left. p, Supranuclear tract (—.—.—.—.) from the left subcortical center (Monakow's) to the homolateral oculomotorius nucleus. q, Supranucleur tract (—.—.—.—.) from the left cortical voluntary center (Monakow's) to the homolateral abducens nucleus. r, A double sided lesion at r would produce paralysis of voluntary movements of the eyes, which at the same time would leave vestibular nystagmus undisturbed (pseudo-ophthalmoplegia of Wernicke). s, A double sided lesion at s would produce paralysis of voluntary movements of the eyes and make impossible the quicker voluntary movements of nystagmus, but would leave the slow vestibular reflex movement undisturbed (supranuclear ophthalmoplegia of Barany).

A further illustration of the voluntary nature of the quick movement of the nystagmus is demonstrated by the fact that the nystagmus is least pronounced when the patient looks toward the side of the deviation and most pronounced when he looks toward the side of his nystagmus.

An interesting and instructive case cited by Barany (Phys. u. Path. der Bgg. Appar. beim Menchen), also seen by the writer, practically settles the problem of the physiology of the two movements of nystagmus. The case is as follows:

A patient was referred from the neurological to the ear department of the general hospital in Vienna, suffering with complete paralysis of abduction and adduction. The patient could not *voluntarily* move the eyes to either side, because of a supranuclear lesion; but upon rotation and caloric irritation of the semicircular canals the patient manifested a reflex conjugate deviation of the eyes to the side opposite the irritation, *without* nystagmus. After turning there was conjugate deviation of the eyes to the same side *without* nystagmus. The writer might cite many other circumstances, including experiments made upon narcotized and comatose subjects, to prove the facts originally stated that the deviation of the eyes or its equivalent, the slow movement of the nystagmus, is the actual vestibular reflex, while the quick movement in the contrary direction is voluntarily produced.

From what has been previously said, the intensity of the vestibular nystagmus depends upon two factors: (1) the intensity of the irritation of the semicircular canals in producing the reflex deviation of the eyes; and (2) the intensity of the voluntary cortical innervation to re-establish macular fixation of external objects. For these reasons the intensity of the nystagmus should be (exactly as we find it) most marked when looking in the direction away from the reflex deviation (toward the nystagmus), and least marked when looking in the direction toward the reflex deviation (away from the nystagmus).

ESTIMATION OF THE INTENSITY OF THE NYSTAGMUS.

The intensity of the nystagmus is estimated by the length of excursions of the eyes and the frequency of these excursions. Thus a nystagmus of six mm. excursion is more intense than one of two mm. Again, a frequency of six excursions per second is more intense than one of two excursions. For the reason that the rapidity of the movements is easier to estimate than the length of the excursions the custom

has been to judge the intensity by this factor; however, the amount of deviation of the eyes measured in degrees would be a more exact but a less practical method.

HOW SHOULD ONE EXAMINE FOR NYSTAGMUS?

The observation of nystagmus is quite important. Low degrees of rotatory nystagmus can be very easily overlooked by casual observation of the eyes between the normal palpebral fissure, and the more so with ordinary illumination.

The writer has described the methods of examination in a former paper upon "Labyrinth Suppuration; Two Cases."

The first requisite is good illumination, which can be obtained by the use of any ordinary head mirror. It is well to direct the illumination sufficiently obliquely (from the side) to avoid too great irritation of the macular region of the eye. This form of illumination serves another purpose, that of producing a bright reflex on the upper part of the sclera. That one may observe the eye movements to the best advantage it is necessary to elevate the upper lid, preferably with the thumb of the left hand when examining the right eye or the thumb of the right hand when observing the left eye. Have the patient look at some distant object in the horizontal plane or slightly above it and directly in front of him. If nystagmus be present, the eye movements can be noted by change of relative position between the bright reflex produced by your concentrated light on the sclera and one of the nearby deep conjunctival or long ciliary vessels which run meridianally. It is also important to look long enough, for in case of late labyrinth suppuration the nystagmus is oftimes so slight that the length of excursions is but one or two mm. at most, and the movements may repeat themselves at intervals of five or six seconds or even longer.

The object of having the patient look at distant objects has been pointed out by Abels and quoted by the writer in former papers upon the subject. When looking at objects at close range the necessary convergence of the eyes calls for so strong a voluntary innervation of the muscles supplied by the oculomotori nuclei and nerves, that it interferes and inhibits considerably the vestibular reflex; again, since the nystagmus is diminished in proportion to the convergence, the same patient with the same amount of irritation in the semicircular canals may at one time show more pronounced reaction than at subsequent times when the oculomotori were more strongly innervated. This may

in a measure account for some of the wide discrepancies in Barany's earlier figures, especially in his observations of after-turning uystagmus. For the same reason the observation of nystagmus by directing the patient to look at the finger held to the sides is a less certain method than the observation of nystagmus after the manner described by the author. However, in some exceptional cases the examination after this coarser method (by having the patient to look to the side) may be permissible, especially in demonstration work before student bodies.

TURNING AND AFTER-TURNING NYSTAGMUS.

THUS far we have observed that vestibular nystagmus is rhythmic in character; that it is designated horizontal, rotatory, vertical, etc., according to the plane of its movements; to the right, to the left, etc., according to the direction of the quicker excursion. We have further observed that vestibular nystagmus can be produced by a one-sided destruction or irritation of certain end-organs in the semicircular canals, irritation of which may be produced mechanically (Ewald's experiments) by turning, by thermic changes and by galvanism.

Of mechanical irritation we have already spoken in the last paper. In the present paper we shall discuss the subject of irritation by turning and after-turning.

Physiologically every act of turning, no matter how slight, is accompanied by a definite amount of nystagmus; for instance, when one turns the head but 20 degrees, the eyes make a double movement; first a slow one, in the direction contrary to the head movement, followed promptly by a second quicker movement in the same direction.

The slow movement is due to the vestibular reflex which permits a brief lingering of the visual fixation upon the object last seen, while the quick movement is due to a second fixation upon a fresh object. The quicker eye movement subtends an arc equal to that of the head movement.

If instead of stopping at 20 degrees, the head is continued in rotation for a total of 360 degrees (thus completing the circle) it will be found that instead of a single slow and quick movement, as in the above instance, the eyes will have made a succession of such movements. In other words, the subject will have manifested a rhythmic nystagmus in the direction of rotation.

The total amount of the quick eye movements equals the total amount of rotation of the head. During each slow movement there is a visual impression together with a fixation of the field, which appears to be constantly passing and which the eyes follow in the direction contrary to the rotation of the head. During the quicker movements there is neither visual impression nor fixation of the field, but a series of interruptions between succeeding visual impressions. In brief,

rhythmic nystagmus is accompanied by a series of alternate visual impressions and interruptions of these impressions.

Thus far we have spoken of the nystagmus produced by rotation of the head with the eyes open, the effects being quite similar but not identical to those produced by the rotation of the field about the stationary subject, the difference being, in the case of rotation of the field about the stationary subject the production of rotational optical nystagmus, while in the case of rotation of the subject with open eyes about the stationary field, the production of combined rotational *optical* nystagmus and rotational *vestibular* nystagmus.

In order to produce pure rotational or turning vestibular nystagmus it is necessary to make the turning with the eyes closed, whereby the optical factor is eliminated.

The demonstration of turning *vestibular* nystagmus can be made by feeling the eye movements thru the closed lids with the finger tips; an experiment made by Kreidl and others which can be repeated easily by anyone upon himself. To examine another person it would be necessary to have a revolving platform sufficiently large to accommodate both the subject and the observer. For practical purposes, however, this method has become obsolete.

With every movement of the head, with closed eyes, there occurs the same movements of the eyes as when the eyes are open; the physiology however differs as has been stated. This then leads us to a discussion of the

PHYSIOLOGY OF PURE VESTIBULAR NYSTAGMUS PRODUCED BY ROTATION.

When the head is erect and the subject turned around the vertical axis with the eyes closed, there is a horizontal nystagmus in the direction of turning which may be felt thru the closed lids; the rapidity of this nystagmus is in proportion to the rapidity of the turning. If the turning is continued long enough, however, the nystagmus ceases. The nystagmus thus produced is known as turning vestibular nystagmus.

If after more or less turning the subject is brought to a standstill, he exhibits an after-turning nystagmus directed to the opposite side to which the turnings had been made; this is designated as an after-nystagmus (a term meaning the same and contracted from the more compound term of after-turning nystagmus). The explanation for turning and after-turning nystagmus offered by Breuer and generally accepted, is that of endolymph pressure against the cupola of the crista

ampullaris which causes an inclination of the cilia, which in turn produces the sensation of turning and the reflex nystagmus. For instance, in the case of turning with the head erect: at the beginning of turning, say to the right, the endolymph in the horizontal canals lags behind because of inertia, just as one standing in a car tends to fall backward when the car starts forward. This results in a relative movement of the endolymph in the horizontal canals in the opposite direction (to the left), which is equivalent to a movement of the endolymph in the right horizontal canal toward the ampulla with a corresponding inclination of the cilia toward the utriculus, and a movement of the endolymph in the left horizontal canal toward the nonampullar end with an inclination of the cilia away from the utriculus.

In both cases—endolymph pressure in the right canal toward the ampullar end and in the left canal away from the ampullar end—nystagmus to the right is produced. These results correspond exactly with the results obtained by Ewald who produced mechanically these same endolymph pressures toward and away from the ampulla.

Thus we see that no matter how it may be produced endolymph motion in the horizontal canal toward the ampullar end causes horizontal nystagmus to the same side, and endolymph motion away from the ampullar, *i. e.*, toward the nonampullar end causes horizontal nystagmus to the opposite side. See illustration, No. X.

This movement of the endolymph in the opposite direction to the movement of the canal continues only so long as there is a c c e l e r a t i o n of the turning motion. Let the turnings become of uniform velocity then the endolymph will sooner or later acquire the same velocity as the canals themselves when there will cease to be any relative movement of the endolymph in the canals. At this moment the cilia according to Breuer and Barany begin to re-establish their normal position of rest—that is a position vertical to their insertion base. This re-establishment to the primary position is brought about by the elasticity of the cilia and of the gelatinous substance which holds them together. This assumption of Breuer, supported by Barany, is based upon the fact that after turning has been continued uniformly for some length of time, the physiological reflex (nystagmus) ceases.

After-turning nystagmus, according to the same authority, is explained as follows:

Taking the same case (subject turning to the right with head erect), at the moment of cessation of turning the canals cease also, but not so

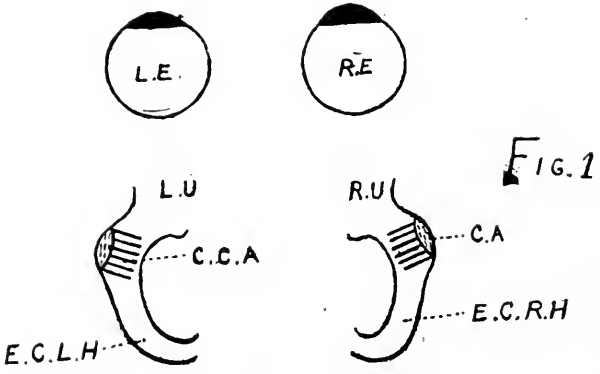


FIG. 1

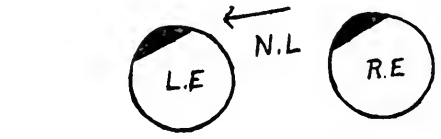
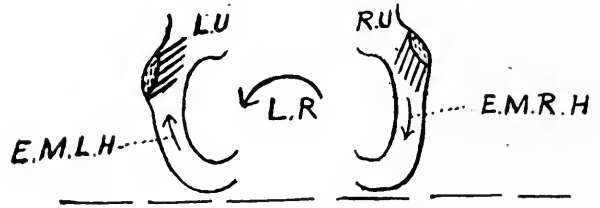


FIG. 2



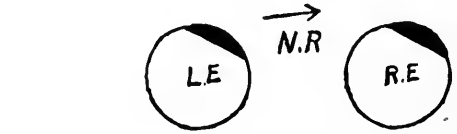


FIG. 3

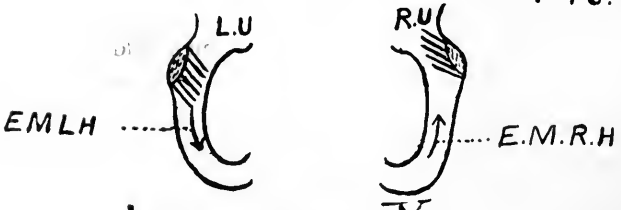


Illustration X

Illustration X. Fig. 1 illustrates the quiescent state of the endolymph in the horizontal semicircular canals. E. C. L. H. represents the external crus of the left horizontal canal; E. C. R. H. the external crus of the right horizontal canal; C. A. the crista ampullaris; C. C. A. the cilia of the crista ampullaris in a state of rest perpendicular to the base. L. U. relative position of the left utriculus; R. U. right utriculus; L. E. left eye, and R. E. right eye; both eyes looking straight ahead.

Fig. 2 illustrates the influence of rotation of the head to the left: (a) Upon the cilia of the crista ampullaris in both right and left horizontal canals, and (b) upon the eyes to produce turning—nystagmus to the left.

The large arrow L. R. represents left rotation of the head. The arrow E. M. L. H. the direction of endolymph motion in the left horizontal canal, toward the ampulla producing an inclination of the cilia toward the left utriculus (L. U.). The arrow E. M. R. H. represents the direction of the endolymph motion in the right horizontal canal away from the ampulla, causing an inclination of the cilia away from the right utriculus (R. U.). The straight arrow N. L. represents the direction of the nystagmus to the left, produced by the inclination of the cilia in either or both canals.

Fig. 3 illustrates the influence of cessation of head rotation to the left: (a) Upon the cilia of the crista ampullaris in both right and left horizontal semicircular canals, (b) upon the eyes to produce after-turning nystagmus to the right. The arrow E. M. L. H. the direction of endolymph motion in the left horizontal canal away from the ampulla, producing inclination of the cilia away from the left utriculus (L. U.). The arrow E. M. R. H. the direction of the endolymph motion in the right horizontal canal toward the ampulla, producing inclination of the cilia toward the right utriculus (R. U.). The straight arrow N. R. represents the direction of the nystagmus to the right produced by the inclination of the cilia in either or both canals.

the endolymph, which continues to move in the same direction, just as one standing in the car tends to move forward for a moment after the car comes to a stop. In consequence there is a relative movement of the endolymph in the right horizontal canal from the ampullar toward the nonampullar end and with it a corresponding motion of the cupula together with inclination of the cilia *away* from the utriculus; and in the left horizontal canal a movement of the endolymph from the nonampullar toward the ampullar end, with a corresponding motion of the cupula and inclination of the cilia *toward* the utriculus; both of which produce an after-turning nystagmus to the left (in the direction contrary to the turning.) The rapidity of the after-turning nystagmus is more or less in proportion to the rapidity of turning which precedes it; however, the duration does not vary appreciably one way or the other.

When the head is held in such a position that rotation occurs exactly in the plane of the horizontal canals, the maximum amount of endolymph motion occurs in these canals (both during a nd after rotation); in which case we have the most pronounced and the purest form of horizontal nystagmus. When the head is inclined away from this plane the amount of endolymph motion in these canals diminishes proportionately, while at the same time the endolymph motion begins to occur in the remaining canals and proportionately to the inclination. As a result, the nystagmus begins to lose its horizontal and assumes another character. If the head is inclined 90° forward or backward, so that the plane of the horizontal canals is at right angles to the plane of rotation, then no endolymph motion in the horizontal canals can occur, but instead endolymph motion in the two vertical canals of each side resulting in the purest form of rotatory nystagmus. The rotatory nystagmus has occurred as a result of the irritation of four vertical canals, two on each side.

Altho Ewald, by careful research with his pneumatic hammer, had determined positively the effects of endolymph motions (both toward and away from the ampulla) in the case of each of the canals it becomes quite a mathematic problem to figure out accurately in which canals the endolymph movement occurs and the direction it occurs in the case of turning in the various planes (frontal, saggital, oblique, etc.). Fortunately, for practical purposes alone, it is not necessary to resort to mathematics, since we have other definite and much simpler laws, to guide us as to the plane and direction in which a nystagmus should occur from turning. They are:

I. Turning nystagmus occurs in the plane of rotation of the head.

II. The direction of turning nystagmus is in the direction of rotation of the head.

III. The plane of the after-nystagmus is the same as that of the turning nystagmus.

IV. The direction of the after-turning nystagmus is opposite to that of the turning nystagmus.

ILLUSTRATIONS.

I. As previously stated, if the head is held erect, so that the horizontal canals lie approximately in the horizontal plane and the subject is turned about the vertical axis to the right (as the hands of a watch move) a rhythmic horizontal nystagmus to the right occurs during the turning and a rhythmic horizontal nystagmus to the left occurs after cessation of turning.

II. If the head is inclined 90° forward so that a plane thru the corneal limbus lies in the horizontal plane and the subject is turned about the vertical axis to the right, a rhythmic rotatory nystagmus to the right occurs during turning to the right and a rhythmic rotatory nystagmus to the left occurs after cessation.

III. If the head is inclined 90° backward so that a plane thru the corneal limbus lies in the horizontal plane and the subject is turned to the right, a rotatory nystagmus to the left is produced during the turning and to the right after cessation.

IV. If the head is inclined 90° to the right so that the vertical meridians of the eyes lie in the horizontal plane and the subject is turned to the right, a vertical nystagmus upward occurs during the turning and downward after turning.

V. If the head is inclined 45° forward (a position midway between horizontal and that of 90° forward) and the subject is turned to the right, a combination of horizontal and rotatory nystagmus to the right occurs during turning and to the left after turning.

VI. If the head is inclined 45° backward (a position midway between the horizontal and that of 90° backward) and the subject is turned to the right, a combination of horizontal nystagmus to the right with rotatory nystagmus to the left occurs during the turning and a horizontal nystagmus to the left combined with rotatory nystagmus to the right after turning.

VII. If the head is inclined 45° to the right and the subject is

turned to the right, an oblique nystagmus upward and to the right occurs during turning and an oblique nystagmus downward and to the left after turning.

From these seven illustrations it is an easy matter to reason out others. All of these forms of nystagmus are increased in intensity when looking to the side toward the direction of the nystagmus and diminished when looking to the opposite side. Following the same law, the case of illustration VI (head inclined backward 45°), resulting in after-nystagmus of combined horizontal to the left with rotatory to the right —the horizontal character is more pronounced when looking to the left and the rotatory character more pronounced when looking to the right.

Vestibular nystagmus is accompanied by other phenomena, both subjective and objective, which may be studied more accurately and to the best advantage when it has been produced by the turning method These are:

I. Visual sensations.

II. Subjective sensation of motion (turning, falling, etc.).

III. Reaction movements. (Equilibrium disturbance.)

The first and second of these may be considered likewise the subjective sensations of vestibular vertigo.

Let us take the case (in which by turning to the left with head inclined forward 90°) a rotatory after-nystagmus to the right has been produced. The rotatory after-nystagmus to the right remains as such so long as the reaction lasts, no matter to which position the head may be changed; however, a change of position of the head will change relatively the plane and direction the nystagmus makes to the erect body and to the external world.

For instance, so long as the head is allowed to remain in the primary position, *i. e.*, head inclined 90° forward to the erect body, so that the face looks toward the floor, the rotatory after-nystagmus to the right is occurring in the horizontal plane and is directed to the right. The quick movements of the eyes occur in the same plane and direction as move the hands of a watch lying upon a table with the face up. The nystagmus is taking place, in relation to the head, in the frontal plane: but in relation to the erect body and also to the external world in the horizontal plane.

If the head of the subject, who remains sitting or standing vertically erect, is shifted to the erect position so that the face is

directed straight ahead, the rotatory after-nystagmus to the right will be found to remain unchanged in relation to the head (frontal plane), but in the relation to the body and to the external world it will have changed from the horizontal (as in the former instance) to the frontal.

If during the reaction the head is turned 90° to the right so that the eyes are directed horizontally over the right shoulder, the rotatory after-nystagmus to the right must occur in the sagittal plane of the body while the quick movement of the eyes will be directed backward. If during the reaction the head is turned 90° to the left so that the eyes are directed horizontally over the left shoulder, then the rotatory after-nystagmus to the right must occur in the sagittal plane of the body while the quick movements of the eyes will be directed forward.

If during the reaction, the erect head is turned 90° backward so that the face is directed towards the zenith, then the rotatory after-nystagmus to the right occurs in the horizontal plane and the quick movements are directed to the left (the same plane and direction as the hands of a watch move when its face is turned downward upon the table or the reverse of that which is made when the face of the subject is directed, downward, toward the floor).

From the foregoing certain facts have been brought out which are common to every after-turning nystagmus and may be briefly stated as:

(a) The plane and direction of after-turning nystagmus retains its same plane and direction relatively to the head as long as the reaction lasts, no matter how the position of the head may be changed.

(b) The plane and direction of the after-turning nystagmus changes its plane and direction relatively to the body and to the external surroundings with every change of position of the head.

These facts must be reckoned with also in considering the three associated phenomena of vestibular nystagmus. It will be found that change of position of the head influences not only the plane and direction of the nystagmus in relation to the body and the external world, as already pointed out, but also the visual sensations, the subjective sensation of motion and the objective (reaction movements) equilibrium disturbance.

I. VISUAL SENSATIONS.

The visual sensations accompanying vestibular nystagmus are expressed variously by different individuals. For instance in the case of rotatory nystagmus to the right, we may by inquiry elicit the following expressions:

(a) The (visual) sensation of objects, rolling *continuously* to the right, *i. e.*, the outer world appears to be rotating about the visual axis to the right.

(b) The sensation of external objects rolling to the right with periods of brief interruptions which find the objects back to their primary (normal) position from which they repeat the rolling.

(c) The sensation of seeing the external world or room double; one stationary and in normal position while the second appears to be rolling to the right.

(d) Some few subjects perceive no motion at all but simply see objects double.

(e) Of those who experience the sensation of external objects moving to the left, the writer doubts the accuracy of their observations.

The sensation of objects rolling to the right, either continuously or, more accurately, interruptedly—as experienced by the vast majority of people during rotatory nystagmus to the right—is the normal sensation and can be easily explained. For instance, an *involuntary* movement of the eyeballs to one side is interpreted as the sensation of motion of external objects to the opposite side; objects to the left are perceived upon the retina to the right of the macula, and conversely every retinal impression which falls upon the left side of the macula is projected to the right and perceived as an object upon the right.

In the case of very active nystagmus of long excursions, the *quick* movements may occur so quickly that retinal impressions are barely possible, but interruptions take place. The quick excursion, however, has brought the eyes back to their primary (normal) position from whence the slow movement begins to repeat itself and with it comes a repetition of the sensation of external objects rolling.

Since retinal impressions are liable to take place during the slow excursion only, then it must follow according to the law of projection that external objects are perceived by the subject as moving in the direction contrary to the slow excursion, in other words, in the same direction as the nystagmus. For illustration see Fig. 2.

In those cases where the subjects express themselves with having seen two rooms, one stationary and the other rolling, the sensations may be explained as follows: that seen as the stationary room in its normal position and relation is due to the *rapid* succession of retinal impressions obtained at the moment the eyes have leaped back to their primary position (Einstellung), which impressions linger sufficiently

long to produce, by their very rapidity, quite a continuous visual sensation. The other or second impression, that of the rolling room, has already been explained.

In the cases where the subjects experienced diplopia without the sensation of objects rolling was due, in the one case seen by the writer, to an actual convergent strabismus which lasted as long as the average after-nystagmus lasts. The eye of the side to which the after-

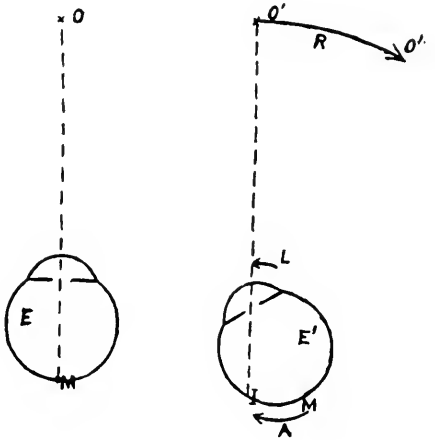


Fig 2

FIG. 2.—E represents the eyeball at rest just before the slow excursion begins. Object O produces a visual image at the macula M and the object is perceived in its normal position. E′ represents the position of the eyeball at the end of the slow excursion to the left, as indicated by the arrow L, the image of object O upon the retina has moved to the left, as indicated by the arrow A to point I. Since retinal impressions at I (to the left of the macula) are referred to the right then the object O has apparently moved to the right, as indicated by the arrow R, to the point O′.

nystagmus should occur was deviated inward. This patient therefore manifested the vestibular reflex (slow movement) only and that of the one eye. The subject having been unable to voluntarily bring the eye back to its primary position (Einstellung) until after the reflex had passed.

Just as in the case of nystagmus so in the case of visual sensations

of external objects rolling, etc., the intensity is increased when looking toward the side to which the nystagmus is directed and diminished when looking toward the opposite side.

Change of position of the head will change the plane and direction of apparent turning. Citation of a single case will be sufficient to illustrate the point.

For instance, in the case of horizontal after-nystagmus to the right so long as the patient holds the head erect he experiences the sensation of surrounding objects rotating in the horizontal plane to the right. If the position of the head is so changed that it is inclined 90° to the right, *i. e.,* the right side of the face resting upon the right shoulder with face directed forward, the horizontal nystagmus will then occur in the vertical sagittal plane of the body and the quick movement will be directed downward; the visual sensation must therefore be that of objects falling vertically downward toward the floor.

II. SUBJECTIVE SENSATION OF MOTION.

The subject with an after-nystagmus experiences a sensation of motion in the plane and direction of his nystagmus which is increased when looking in the direction of the nystagmus and diminished when looking in the opposite direction. For instance, a patient with horizontal nystagmus to the right experiences a sensation of turning or rotation to the right. In case of rotatory nystagmus to the right the plane of the nystagmus is frontal, hence he suffers the sensation of falling laterally to the right. In the case of vertical nystagmus downward, the plane of the nystagmus is sagittal, hence the patient has the sensation of falling forward.

These sensations are not entirely dependent upon the visual impressions, for they are present with the eyes closed as well as with the eyes open. Again the subjective sensation of motion endures as long as the after-nystagmus, and is felt by the subject whether he is standing, sitting or lying down.

The sensation of turning or falling *when the eyes are open* is dependent mostly upon the visual impressions—that of objects turning or tumbling. As the room in which one is standing seems to turn in a given direction the subject will naturally feel as tho he is going with it. This same sensation of turning or tumbling is experienced by one with rhythmic nystagmus even when produced optically, as by Mach's rotating cylinder experiments, in the muscle paresis, etc. Visual im-

pressions however do not explain the sensation of turning or falling experienced in vestibular nystagmus *with the eyes closed.*

Barany (Phys. u. Path. des Bogeng.) who has studied the subject most carefully is undecided as to whether the falling sensation is due to an irritation conveyed directly to the cortex thru vestibular fibers, or whether the sensation arises thru the medium of the nystagmus. According to Ewald (Physiolog. Untersuchungen ueber das Endorgan des N. Octavus), we are led to believe that the sensation of falling is due to an overbalance of muscle innervation of one side of the body produced by the vestibular reflex.

There are certain facts which contradict the theory that these sensations are the result of impulses which are conveyed to the cortex. Flourens (Comptes Rendus, Tome LII, p. 673-75) and others have found that animals whose cerebral hemispheres have been removed show the same reactions as before. Again, if cortical perception was entirely responsible for the falling sensations then the sensations should remain constantly the same in spite of change of position of the head, which is contrary to the facts as we find them.

For similar reason we must deny Ewald's explanation of muscle tonus.

If the muscular hyper- and hypotonus theory was correct then movements of the head should not influence the plane or direction which the sensation of turning or falling takes, or that of the actual reaction falling.

For instance, a patient manifesting a rotatory nystagmus to the right because of destruction of the left semicircular canals or stimulation of the right semicircular canals, the hypertonus of one side and hypotonus of the opposite side should remain constantly the same in spite of any change of position of the head; the subject should fall constantly to the one side. The fact however is that both the sensation and the reaction falling may be reversed in their directions by a change of position of the head of 180°.

From the facts as we find them it would seem that one explanation alone is left us, and that is: The sensation of falling and the objective falling is dependent not upon the cerebral hemispheres, but upon the cerebellum.

The writer would attempt the following explanation: That the reflex slow movement of the eyes to the left in case of stimulation of the right semicircular canals occurs independently of the great brain, as

shown by Flourens's experiments. Furthermore, since we have come to accept the cerebellum as the central organ for the control of equilibrium and that the kinesthetic sense impulses are conveyed to the cerebellum from the deep muscles, joints, etc., from all parts of the body, may we not look upon the reflex contraction of certain eye muscles as producing similar kinesthetic sense impulses which are conveyed likewise to the cerebellum? In brief, the opinion of the writer is that when by stimulation of the right horizontal semicircular canal the eyes deviate to the left, the involuntary reflex contraction of the left abductor and the right adductor causes a kinesthetic sensation of contraction of these muscles which is conveyed to the cerebellum in the same manner as all other kinesthetic sensations are, and is interpreted in the cerebellum as the sensation of torsion of the body to the right which the cerebellum as the central organ for equilibrium attempts to correct by sending motor impulses to certain body muscles to produce the opposite torsion. However, since the sensation of torsion is only apparent and not real, the attempt to unwind the apparent body torsion produces an actual torsion in the opposite direction.

III. REACTION MOVEMENTS (EQUILIBRIUM DISTURBANCE).

The subject with an after-nystagmus will move or fall in the plane of his nystagmus but in the contrary direction. For instance, a subject with horizontal nystagmus to the right will tend to turn in the horizontal plane, that is make a torsion movement of the head and body to the left. In the case of rotatory nystagmus to the right the plane of the nystagmus is frontal, hence the subject will tend to fall laterally to the left. In the case of vertical nystagmus downward the plane of the nystagmus is vertical and the direction is downward, hence the subject will tend to fall vertically backward.

As pointed out elsewhere the plane and direction of an after-turning nystagmus remain the same, no matter how we may change the position of the head. A rotatory after-turning nystagmus to the right remains as such whether the head is kept forward, turned upward, backward or to either side; however, with a change of the head position comes a change in the plane and direction of the falling sensation and of the actual falling.

A few cases may be cited to illustrate this point. For instance, in case of rotatory nystagmus to the right the subject has the subjective sensation of falling laterally to the right, because of which in his at-

tempt to correct he really over corrects and actually falls laterally to the left. Now if the subject's head is turned 90° to the left so that the face is directed to the left, his rotatory nystagmus to the right continues as such, but it occurs in the sagittal plane of his body. As a result the subject experiences the subjective sensation of falling forward, which his attempt to correct causes him to fall backward. If the head should be turned 90° to the right, so that the subject is looking horizontally to the right, the rotatory nystagmus is taking place in the sagittal plane and directed backward and downward, in which case the subject experiences the false subjective sensation of falling backward, which in his attempt to correct causes him to fall forward. If the head should be inclined forward 90° so that the subject looks downward, toward the floor, the rotatory nystagmus to the right occurs in the horizontal plane; the subject experiences the false sensation of torsion to the right, which in his attempt to correct he actually turns around the vertical axis of his body (in the horizontal plane) to the left. In the case of horizontal nystagmus to the right with the head upright the subject naturally suffers the sensation of turning in the horizontal plane to the right, but actually turns to the left. If the head is inclined 90° forward so that the patient is looking toward the floor the horizontal nystagmus remains as such, but the nystagmus occurs in the frontal plane of the body, which is equivalent to a rotatory nystagmus to the left with head erect, the corresponding false sensation is that of falling to the left, which causes the patient to actually fall to the right. If the head is inclined 90° backward so that the subject looks toward the zenith, the horizontal nystagmus remains the same, but the nystagmus occurs in the frontal plane of the body, equivalent to a rotatory nystagmus to the right with the head erect, in which case the patient experiences the false sensation of falling to the right, which results in the subject actually falling to the left. So we might continue the number of illustrations.

Barany teaches (Phys. u. Path. des Bogeng. Appar. beim Menschen, page, 14, third paragraph from the bottom) that the sensation of falling is increased when the eyes are closed. This is probably so, for we find the subject has a greater tendency to fall with the eyes closed than when open.

Beside the above typical phenomena which are constantly present in association with vestibular nystagmus, we find other symptoms more or less present. These are:

IV. Nausea and vomiting, which occur most constantly in connection with the rotatory form of nystagmus and least constantly in connection with the horizontal.

V. Vasomotor changes. Flushing and pallor of the face, profuse sweating, etc.

VI. Nervous and hysterical phenomena and epileptiform attacks are not uncommon in those so inclined.

METHOD OF MAKING THE EXAMINATION.

As a result of his thoro and painstaking experiments, Barany has developed an accurate method for the determination of the reactions of the semicircular canals to turning. At the same time he has supplied us with data and figures of inestimable value in the differentiation of the normal from the abnormal.

Before Barany began his researches, the best apparatus for the study of turning nystagmus was a large revolving platform upon which sat or stood both the subject and the experimenter. This apparatus was cumbersome and costly. At that time the experimenters studied mostly the reactions (nystagmus) *during* the turnings.

Later, as Barany found the reactions *after* turning were just as positive and even more accurate than *during* turning, he discarded the revolving platform, and used the less expensive and more available revolving stool, so that today the method as developed by Barany can be practiced by any one in his office and without the aid of an assistant.

To make the examination, one needs a suitable revolving stool fitted with a bearing that does not permit elevation or depression when turning, and a metal or wooden bar fastened to the back and reaching above the patient's head to serve as a handle. An illustration of the one used by the writer may be seen in the paper upon "Labyrinth Suppuration—Two Cases." Besides this, one needs a stop watch and a pair of opaque spectacles. The spectacles used by the writer are nothing more than a pair of dark glasses with adhesive plaster fastened to the external surface.

The subject should be seated comfortably in the revolving stool with the feet just escaping the floor. It is well to have handles on the chair that the patient may hold on well and avoid toppling over. The examiner stands in front of the chair with his feet and legs far enough from those of the subject to avoid collision. In one hand is held the

stop watch, with the other hand reaching over the subject's head the top of the handle is grasped. The patient is instructed to hold fast for fear of losing his balance. The turnings should be made as nearly uniform as possible, consuming about two seconds for a complete turn (360 degrees). Turning ten times (3,600 degrees) as a rule produces the maximum amount and duration of after-nystagmus, as determined by Barany and corroborated by others. When one wishes to examine for horizontal after-nystagmus to the right, naturally we must turn the subject to the left, *i. e.,* as the hands of a watch move when lying upon the floor with the face down, and vice versa for horizontal after-nystagmus to the left.

If one wishes to examine for rotatory after-turning nystagmus to the right, the head should be inclined 90° forward and the patient turned to the left (as the hands of a watch move lying upon the floor with the face down), and vice versa for rotatory after-turning nystagmus to the left. After ten complete turnings the subject is brought to a sudden stop, when the examiner starts the watch and begins to observe the nystagmus. As soon as the nystagmus ceases, the watch is stopped. If the observations have been correct, the stop watch should record the exact duration of the after-nystagmus. It is well for beginners and of advantage to the expert, providing the patient can tolerate it and does not object, to make a second experiment to corroborate the first findings. It is needless to add, that all experiments and observations should be made in a well lighted room, and if necessary a head mirror should be used by the experimenter to better illuminate the subject's eye.

Since both eyes move in unison, the observation of one eye is generally sufficient to note the reactions to both the right and the left side. It is well, however, since exceptional cases of unilateral nystagmus do occur, to casually observe the fellow eye.

The reaction of the semicircular canals to turning is in proportion to the duration of the after-nystagmus. The average duration of after-turning nystagmus is between twenty and twenty-two seconds for the horizontal without the use of the opaque spectacles, and between twenty-three and twenty-six seconds for the rotatory. With opaque spectacles fitted before the subject's eye the duration of the horizontal after-nystagmus is considerably lengthened and endures on an average from thirty-six to forty seconds.

The examination of horizontal after-nystagmus with the opaque

spectacles is to be preferred to that made without, since Abels has pointed out that convergence of the eyes inhibits the free play of the vestibular reflex. The writer has alluded to this subject before and will not go into further details now. Tho the figures given above represent the average duration of rotatory and horizontal after-nystagmus Barany points out some wide variations that he has found in some of his experiments upon normal people.

In his experiments upon 200 persons, some normal and others with ear affections but not complaining of vertigo, he found the average duration of horizontal after-nystagmus after ten turnings to the left forty-one seconds, and to the right thirty-nine seconds.

These figures are somewhat high, but may be explained as due to the fact that many of those with ear affections may have suffered slight irritation of their labyrinths, which would tend to lengthen the duration of the after-nystagmus.

For the fear of confusing the nystagmus due to the reflex and that "spontaneous nystagmus" met with in most individuals, due to over strain of the extra ocular muscles during attempts to gaze in extreme directions, Barany constructed a special instrument which he calls a "Hilfinstrument zur Beobachtung des rotatorischen Nystagmus" to aid him in his observation of rotatory after-nystagmus; illustration and description of which may be found on page 19 of his booklet. The writer has used this instrument at the request of Barany and discarded it again. Barany himself has discarded it also and brings it out only occasionally to show the students.

I do not consider observations of after-turning nystagmus as thoroly accurate when the subject looks in any other direction than straight ahead. This point has been dwelt upon by me in former papers. I am led to this conclusion by repeated experiments made upon more than 400 subjects. Besides I find that my figures are more uniform and consistent than those of Barany, which I believe is due to the very fact that my observations have been made with the subjects looking straight ahead instead of to the side as Barany's have been made.

I have never been able to find the extremely high figures even in cases of marked labyrinth irritation as Barany has in some of his normal cases (88 and 120 seconds).

According to Barany the duration of the after-nystagmus diminishes slightly after fifty years of age; which corresponds with the results obtained by the writer.

The duration of after-nystagmus varies according to the number of times the subject has been turned. It is proportionately less where the turnings are less than ten, *i. e.,* the duration averages less for two turns than for five, and less for five than for ten turnings.

Again a greater number of turns than ten seem to diminish rather than increase the duration of after-nystagmus. For instance, the average duration of after-turning nystagmus is less after twenty than after ten turnings, and less after forty than after twenty. These data are important since they prompt us to adopt the method of Barany, *i. e.,* to practice ten turning tests rather than more or less than ten.

Normal subjects generally show about the same reaction (duration of after-nystagmus) when examined repeatedly upon different days. However, slight variations have been found by Barany which the writer believes is due rather to his method of examination—having the subjects look laterally—than to an actual variation in the semicircular canals' reactions.

In Austria where people dance continuously to the right or the left, as the case may be, without reversing Barany found the physiological reactions vary somewhat to the two sides. For instance, with those who dance to the right, the duration of after-nystagmus to the right was found to be of slightly shorter duration than the after-nystagmus to the left.

Rapid turnings produce a more rapid nystagmus both during and after turning, but the duration of the after-nystagmus does not vary from that produced by the slower turning. In some cases the duration was longer and in other cases shorter after slow turnings than after quicker turnings; but on the whole they average up about the same.

Barany refers to a form of nystagmus which he terms "nachnach nystagmus" (after-after-nystagmus). It occurs not infrequently in those who have been turned twenty or more times. In those cases the subject shows first a very pronounced after-nystagmus in the usual direction (contrary to the turnings), which ceases rather suddenly, followed by a small excursioned nystagmus in the opposite direction to the after-nystagmus (that is, in the direction of the turnings which produced it); this may last as long as sixty seconds. This form of nystagmus is more of scientific interest than of practical value.

Since the semicircular canals of one side are capable of producing nystagmus to either side it becomes a question of interest and importance to know just how much the after-nystagmus to one side is due to

irritation of the semicircular canals of the same side and how much to irritation of the semicircular canals of the opposite side.

(1) We have learned from Ewald's experiments that mechanical forcing of the endolymph toward the ampulla of the horizontal canal produces nystagmus to the same side; while suction produces the opposite movement of the endolymph and with it the opposite nystagmus.

(2) In a normal individual it is impossible, by turning, to produce endolymph motion of one side only.

(3) In a normal case with an after-turning nystagmus of twenty-four seconds (the approximate normal) duration, it is apparent (from the foregoing) that irritation from the two sides is responsible for the after-nystagmus.

(4) If the same patient should lose the labyrinth of one side from any cause it will be found that the after-nystagmus to the well side will have diminished from twenty-four to sixteen seconds (which represents an average obtained from an examination of numerous cases); while the after-nystagmus to the diseased side will have diminished from twenty-four to approximately eight seconds.

(5) It will be seen from the above figures that in a case of one-sided labyrinth destruction the after-nystagmus to the sound side is twice as long as the after-nystagmus to the diseased side.

(6) The greatest common divisor of the above figures (8, 16, 24) is 8 from which we may simplify the solution of our problem as follows: For instance, in a case of left-sided destruction—

(a) The duration of after-nystagmus to the right, representing irritation of the right side only, is sixteen seconds.

(b) In the same case the duration of after-nystagmus to the left, representing irritation of the opposite side only, is eight seconds or just one-half the value of irritation of the right side.

(c) In the normal case, both sides intact, the duration of the after-nystagmus to the left, representing irritation of the both sides, is twenty-four seconds or sixteen for the same side plus eight for the opposite side making the total of twenty-four seconds. These figures are not to be considered exact for *all* cases, for we find many variations, but in the main they represent a fair average, demonstrating that after-turning nystagmus to one side is due to two irritations, two-thirds of which is produced by irritation of the semicircular canal of the same side and one-third by irritation of the semicircular canal of the opposte side.

CALORIC NYSTAGMUS.

CALORIC Nystagmus is the name given to the rhythmic nystagmus which results from the application of heat or cold directly (thru a perforation in the tympanic membrane) or indirectly (in case of intact membrane) to that portion of the external labyrinth wall which forms the inner wall of the middle ear spaces.

The phenomenon of vertigo after lavage of the middle ear spaces had been observed and reported by numerous authors. Quoting from Barany, pages 26 and 27 of his work—"Phys. u. Path. des Bgg. Appar. beim Menschen":—"Smiegelow and Hensen in the year 1868 discovered, while experimenting upon the strength of resistance of the tympanic membrane, that the influence of cold water in the external canal produced vertigo, nausea and vomiting, while water at the body temperature did not. Other authors have corroborated the fact that syringing the ear with too cold or too hot water produces vertigo (Cohn, Urbantschitsch)."

"Baginsky, in the year 1881, made animal experiments, syringing the ears of rabbits with cold water. He found that it produced the same vertigo and nystagmus, but as soon as warm water was used the vertigo and nystagmus ceased. Baginsky then increased the pressure until the tympanic membrane and the membranes of the round and oval windows were ruptured and water rushed into the semi-circular canals and thence into the brain. Since the animals died of meningitis, Baginsky concluded that the nystagmus was not produced from the semi-circular canals but from the brain itself. He neglected to observe, however, whether with the use of the cold water these membranes were *always* destroyed."

For three years prior to 1907, Barany investigated thoroly the subject of caloric nystagmus, and thru his efforts have been established certain definite facts concerning the reactions of the ear labyrinth to heat and cold. Besides, he has given us a theory which satisfactorily explains all the phenomena of caloric nystagmus. Since his publication (1907) nothing new has been added to this branch of the subject nor has Barany been compelled to retract a single statement. Concerning the theory advanced by Barany a few have ventured to disagree, but without avail. His theory stands today stronger than it did

before these authors attempted to disagree with him. The subject of caloric nystagmus is so linked with Barany's work that it is well nigh impossible to write upon this branch of the subject in anything like original style.

The following are the reactions of the ear labyrinth to heat and cold as determined first by Barany and since corroborated by every investigator.

I, With the head erect, cold water (i. e. water below the body temperature) syringed into the right ear produces a rotatory nystamus to to the left or opposite side.

II, With the head erect, hot water (i. e. water above the body temperature) syringed into the right ear produces a rotatory nystagmus to the right or same side.

III, With the head inverted (i. e. vertex toward the floor) cold water syringed into the right ear produces rotatory nystagmus to the right or same side.

IV, With the head inverted, hot water syringed into the right ear produces rotatory nystagmus to the left or opposite side.

V, With the head inclined to the left so that a line uniting the two eyes stands vertically, cold water syringed into the right ear produces a horizontal nystagmus to the right or same side.

VI, With the head inclined to the right, cold water syringed into the right ear produces a horizontal nystagmus to the left or opposite side.

VII, With the head inclined to the left, warm water syringed into the right ear produces a horizontal nystagmus to the left or opposite side.

VIII, With the head inclined to the right, warm water syringed into the right ear produces a horizontal nystagmus to the right or same side.

The writer was the first to observe a mixed reaction from the use of cold water syringed into the ear, as follows: with the head inclined obliquely to the left, cold water syringed into the right ear will produce a pronounced horizontal nystagmus to the right side combined with a moderate degree of rotatory nystagmus to the left. In order to obtain this reaction, it is not necessary to have the head held exactly at an angle of 45° to the left, for the horizontal feature will be manifested even with a much less inclination of the head. I cite this reaction for the purpose of avoiding confusion to anyone who may see it

for the first time. It comes natural to one in syringing an ear to have the patient's head inclined somewhat away from the operator introducing the canula, so the first evidence of a positive reaction may be a pronounced horizontal nystagmus to the same side. Change of position of the head to the erect causes the nystagmus to change to the typical rotatory character to the opposite side.

That the horizontal element predominates in the case of the mixed nystagmus referred to is due probably to the fact that the horizontal semicircular canal is more exposed to the influences of temperature changes than the superior vertical canal.

It has been observed that in the case of after-turning nystagmus, the plane and direction of the nystagmus when once begun remain unaltered by any subsequent change of position of the head. In the case of Caloric Nystagmus the opposite condition holds true, viz.—a subsequent change of position may alter either the plane or direction or both plane and direction of the nystagmus. This characteristic is a distinctive feature of the Caloric Nystagmus.

In the case of Galvanic Nystagmus, the polarity determines the direction and no change of position of the head will change either the character or the direction of the nystagmus.

THE THEORY OF BARANY.

Briefly put, the theory of Barany is as follows:—endolymph motion in the semicircular canals is produced by an increase in density of the fluid in that part of the canals exposed to cold, causing it to sink; and by the diminution of the density of the fluid in that part of the canal exposed to heat, causing it to rise. Let the labyrinth be represented by a vessel filled with a fluid of even temperature (37° C.) upon the external surface of which is played a stream of cold water (Fig. 1). The fluid immediately behind the exposed external wall is cooled, causing it to sink to the deepest part of the vessel, while the uncooled water above moves downward to take its place. The result is a circulation or tendency to circulation of the fluid in the vessel. It is evident that hot water would produce the opposite effect to that produced by cold water.

Altho the density of the endolymph increases when the temperature is reduced below 37° C. (body temperature), we see in fig. 1 that the arrow indicating the direction of the cooled endolymph next to the

wall c is downward in both instances (A, vessel upright and B, vessel inverted), but in relation to the surfaces of the vessels the direction in the case of B (vessel inverted) is the reverse of that in the case of A (vessel erect).

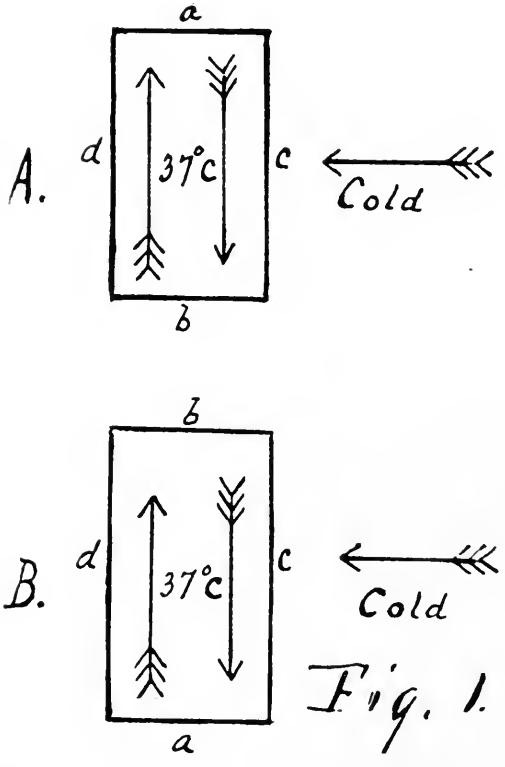


FIG. 1.—Schematic representation of the vestibular apparatus by a vessel filled with a fluid at a temperature of 37° C. (98.6° F.); after Barany.

A. The direction of the fluid circulation when the wall c is cooled with a stream of cold water. The vessel is shown in the upright position. The surface (a) indicating the top and the surface (b) indicating the bottom of the vessel.

B. The direction of fluid circulation when the wall c is cooled with a stream of cold water. The vessel is here indicated in the *inverted position;* the bottom (b) is above and the top (a) is below.

In order to fix more closely in the mind the caloric reactions, the writer has made use of two other illustrations—figures 2 and 3.

In figure 2 is represented the right membranous labyrinth viewed

from the external surface with the head erect. In this position the external canal lies approximately in the horizontal plane, hence the application of heat or cold produces but little or no effect upon the endolymph contained within it. The superior canal, on the contrary, lies approximately in a vertical plane and the endolymph within it must necessarily be more subject to upward and downward movements from temperature changes than in the case of the external canal.

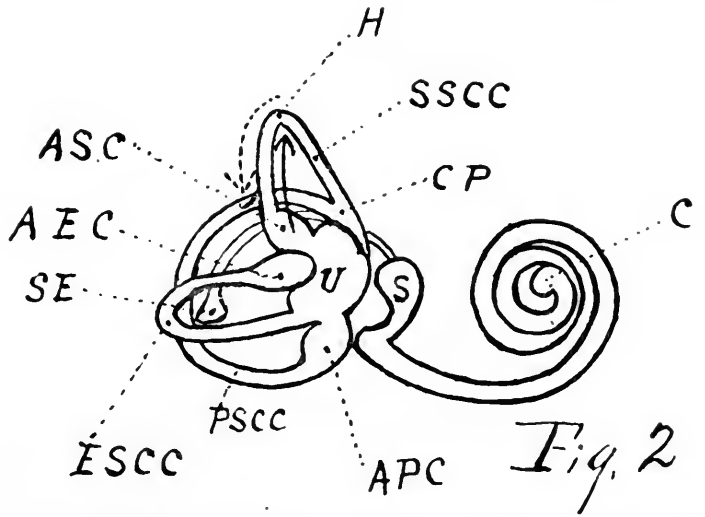


FIG. 2.—Schematic representation of the right membranous labyrinth viewed from the external surface with the subject in the erect position. C, cochlea; S, sacculus; U, urticulus; APC, ampulla of the posterior semicircular canal; PSCC. posterior semicircular canal; ESCC, external semicircular canal; SE, saccus endolymphaticus; AEC, ampulla of the external semicircular canal; ASC, ampulla of the superior semicircular canal; H, highest point of the superior semicircular canal; SSCC, superior semicircular canal; CP, common portal of the superior and posterior semicircular canals. The dotted arrow indicates the direction of endolymph circulation during the application of cold. The full arrow indicates the direction of endolymph circulation during the application of heat.

tion in this direction to the same effects produced by the mechanical

A play of cold water upon the inner wall of the middle ear will cool the endolymph in the utriculus and the external crus of the superior canal. The cooled endolymph sinks in the direction indicated by the dotted arrow. We have but to compare the effect of endolymph mo-

experiments of Ewald or to those produced by turning to the left with head inclined 90° forward to determine the results. In all three cases inclination of the cilia of the crista ampullaris toward the utriculus is produced and with it a rotatory nystagmus in the opposite direction of the inclination (to left side).

From what has already been said, it is evident that the application of heat produces the opposite endolymph motion, indicated by the full arrow, and with it a rotatory nystagmus in the same direction of the inclination (to the right side).

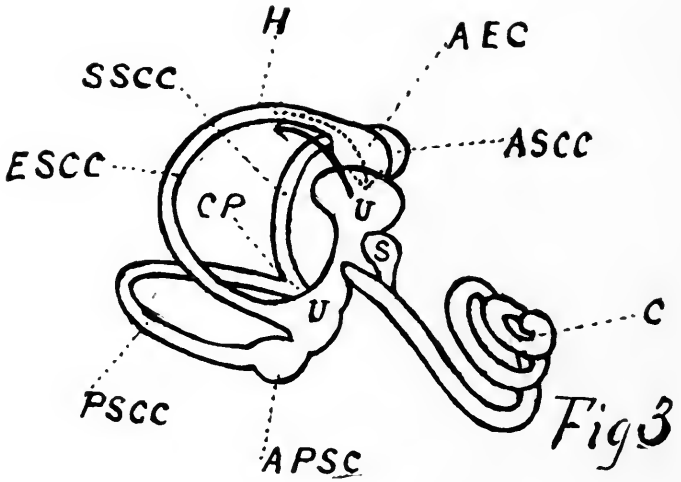


FIG. 3.—Schematic representation of the right membranous labyrinth viewed from below, the position it is in when the head is inclined to left 90° (left side of the face resting upon the left shoulder). C, cochlea; S, sacculus; U, utriculus; A PSC, ampulla of the posterior semicircular canal; P S C C, posterior semicircular canal; E S C C, external semicircular canal; S S C C, superior semicircular canal; H, most superior part of the external semicircular canal; A E C, ampulla of the external semicircular canal; A S C C, ampulla of the superior semicircular canal; C. P., common portal of the superior and posterior semicircular canals. The dotted arrow indicates the direction of endolymph circulation during the application of cold. The full arrow indicates the direction of endolymph circulation during the application of heat.

In figure 3 is represented the right membranous labyrinth in the position it occupies when the head is inclined laterally 90° to the left. The external surface is directed upward. In this position the external

canal lies approximately in a vertical plane and for this reason particularly the application of heat or cold must produce the maximum amount of endolymph motion in this canal. There are other reasons too for this maximum of endolymph motion in the external canal rather than in the superior canal, which also lies in a vertical plane at right angles to the external canal; (1) that the highest point in the external canal is higher than the highest point in the superior canal, and (2) it lies nearer to the point of application of the heat or cold than the superior canal. A play of cold water upon the inner wall of the middle ear will cool the endolymph in the utriculus and the exposed part of the external crus. The cooled endolymph must sink in the direction indicated by the dotted arrow. Again we have but to compare the effects of endolymph motion toward the ampulla and utriculus in the case of the external canal with those produced by Ewald's pneumatic hammer by compression or by turning to the right with the head erect in order to determine the results. All three produce inclination of the cilia of the crista ampullaris toward the utriculus and with it a horizontal nystagmus to the same side (right).

If the play of cold water upon the labyrinth, with the head in this position, produces horizontal nystagmus to the same side then it follows that hot water must produce the opposite effect, viz.—endolymph motion in the horizontal canal in the opposite direction indicated by the full arrow and with it a horizontal nystagmus to the opposstte side (left).

Opposite effects to those enumerated are produced by change of position of the head 180°. Furthermore, the direction and plane of the nystagmus may be definitely changed by altering the position of the head even after the reaction has once begun. This feature is characteristic alone for this form of irritation which was alluded to earlier in the paper and is one of sufficient importance to bear this reiteration.

The caloric reaction of the labyrinth is positive when nystagmus results and negative when nystagmus does not result. We therefore find the reaction positive in normal labyrinths and all pathologic conditions which fall short of complete destruction. We find the reaction negative in complete destruction of the labyrinth and complete disability of the nerve, be it temporary or permanent. But one thing it does not tell and that is the intensity of an existing pathologic process in the labyrinth when that process is other than a complete destruction.

In other words, the caloric test, altho a perfectly satisfactory qualitative test, is in no way a quantitative one like the turning and galvanic tests.

METHOD OF MAKING THE CALORIC TEST.

The caloric test may be made with either water or air injected into the canal or better, when possible, directly into the tympanic cavity.

Since water is capable of carrying a greater number of heat units to and from the parts than air or vapors, it is naturally better suited for the purpose. In fact, for a patient to react at all promptly to the caloric test with the use of air or vapors it would be necessary to have a suitable generator, the simplest of which would necessarily be quite complex in structure. I would hardly advise the use of ethyl chloride vapor since it is impossible to regulate the temperature satisfactorily and harm can be done. In fact I have never made a test with air or vapor nor have I seen or read of anyone else making any. The investigations of Barany, Alexander, Neumann, Ruttin and myself have all been made with water, which is quite simple to make and the most satisfactory.

The selection of the temperature of the water to be used. Remembering that the temperature of the endolymph in the canals is about that of the body (between 98.6° and 100° F.), it is evident that in order to obtain a positive reaction from heat we must use water the temperature of which is at least 10 degrees higher. This temperature can be borne comfortably by the patient. An increase of 10 degrees more, however, is about the limit which the patient can tolerate. A difference of 20 degrees then is about the limit of high temperature for water to be used in making a caloric examination. This difference is sufficient to produce a caloric reaction under favorable circumstances; i. e., large perforation in the tympanic membrane, the absence of excessive granulations or cholesteatomatous masses, marked thickening of the mucous membrane, acute inflammation or acute exacerbation of a chronic inflammation or high fever.

Alexander pointed out the importance of fever upon this reaction. A patient suffering from a chronic middle ear suppuration with a large perforation and w i t h o u t f e v e r may react promptly today with water at the temperature of 118° F., but tomorrow with the patient suffering fever (104°, 105° or 106°), water of 118° may fail to produce a positive reaction while the labyrinth condition has remained unchanged.

The use of hot water is not so trustworthy for testing the labyrinth-

ine function as is the use of cold water. The cold water is by far the more satisfactory, and is used almost exclusively by all investigators, the reason being that a greater variation of temperature from the normal (98.6° F.) may be applied without any discomfort to the patient. Water at the temperature of 68° can be comfortably borne by the patient; besides, the density of the endolymph is considerably influenced by this difference (30°) of temperature. Lower temperatures may however be employed and tolerated, but these lower temperatures are not necessary except in a small percentage of cases presenting certain obstacles: atresia of the canal, acute narrowing of its lumen, foreign bodies in the canal, tumors, intact membrane, small perforations, acute inflammation of the tympanic cavity, excessive amount of granulations or cholesteatomatous masses.

The use of water in the ear is generally contra-indicated in cases of healed middle ear suppuration with remaining dry perforation.

METHOD OF APPLYING THE TEST.

Before beginning the test it is necessary to note the presence or absence of spontaneous nystagmus, and when present its character, plane and direction. Ascertain too the positions of the eyeballs at which the spontaneous nystagmus is most and least pronounced. It is well to have one observer to direct the gaze of the patient and observe the reaction. The finger of the observer may be used for the purpose or a specially designed instrument of Barany known as the Hilfsinstrument, or, better still, have the patient look at a d i s t a n t point (say a nail in the wall). A second operator applies the stream of water into the canal or better, where possible, directly into the tympanic cavity. The instrument used in the Politzer and Alexander clinics and by the writer, consists of a Politzer bag attached to a soft rubber tube about one foot long and on the distal end of this tube is fitted a Hartman's attic canula. The safest and surest method of introducing the canula properly is with the aid of a speculum and head mirror. The introduction and holding of the speculum in place occupies one hand of the second operator and the introduction of the canula his other hand. Someone else (a third party) must elevate and squeeze the Politzer bag containing the water. Since this requires no especial skill, it may be done by a nurse or friend accompanying the patient.

I have, however, occasionally managed without any assistant, but it is never so satisfactory as when you have one and better when you have two.

It is especially desirable to have one trained assistant at least, to either observe the first signs of reaction (nystagmus) while you introduce the canula or vice versa. Since the caloric reaction is sometimes quite violent and unpleasant, it is well to note the reaction directly it is manifested and then be prepared to control it promptly. As soon as the reaction is sufficiently pronounced to be sure of its presence, immediately detach the balloon containing the cold water and substitute for it one containing warm water. The play of warm water soon counteracts the effects of the cold.

This is the practice of the writer and one which he strongly recommends for the comfort of the patient.

At this juncture I would further suggest that directly you have observed the cold water reaction (rotatory nystagmus to the opposite side) that you direct the patient to look to the side away from his nystagmus in order to diminish the intensity of the accompanying vertigo along with its train of unpleasant sensations.

In making the cold water test the first sign of reaction may be the gradual increasing of an existing spontaneous nystagmus, or it may be the beginning of a nystagmus which had not existed spontaneously.

After withholding the cold water it will be seen that the nystagmus which began quite mildly at first increases in intensity until it reaches its maximum. This occurs frequently as late as 30 seconds after the discontinuance of the cold water. It is for this reason that I adopted early the plan referred to above, of counteracting the effects of cold water with the immediate use of warm water.

The examination of the caloric irritability of the labyrinth may be conducted with the patient sitting up or lying down. It is generally more satisfactory to examine the patient while sitting up. The examination of the patient while lying in bed is very unhandy, since a portable light is necessary for illumination and considerable care must be exercised to avoid getting the bed wet. These obstacles however can be more or less overcome or overlooked by an examiner who is bent upon his task. There are certain cases where the patient's physicial condition will not permit the test being made in the sitting posture. Take, for instance, a patient in a comatose or semicomatose condition; for this character of case the examination in bed will be found to be especially suited.

Concerning the examination of these cases, we must not forget that a positive reaction in a comatose

patient is manifested by a conjugate deviation of the eyes in the direction contrary to that of the nystagmus which the patient would have shown had he been normally conscious.

There are some obstacles to the carrying out of the caloric test in some cases, among which may be mentioned:—

I, A completely occluded canal from (a) congenital atresia; (b) the presence of a large foreign body; (c) the presence of a large furuncle in the cartilaginous canal; (d) the presence of a periosteal or subperiosteal swelling in the osseous canal in cases of mastoid abscess; (e) exostoses, tumors, etc. In these cases it is often impossible to bring the water near enough to the parts to produce effect.

II, The presence of excessive granulations springing from the tympanic cavity, reaching well into the external canal, may prevent or hinder the entrance of water into the tympanic cavity. In other less pronounced cases the position of the polyps may hinder the water from reaching the inner wall of the middle ear and thus at least delay the reaction. It is well in these cases to use water of a lower temperature and for a longer period (3 to 5 minutes). Sometimes a pronounced reaction may be obtained after three or five minutes when a shorter length of time would have failed completely.

III. Cholesteatoma, since it breaks up readily under the influence of a fairly continuous stream, seldom presents an obstacle sufficient to prevent a reaction; but it frequently delays it.

IV, Acutely inflamed tympanic cavity with a red swollen membrane either intact or with a small perforation delays the reaction considerably and an unpersistent examiner may after two minutes' trial either give up or decide that the labyrinth is nonreactive, whereas a second trial with colder water used over a longer period may succeed in bringing about a typically positive reaction.

There is one caution that might be suggested to a beginner and that is to be careful in every case with a normal membrane. The writer saw two cases of middle ear suppuration produced by carelessness on the part of students in the introduction of the canula. The complication was the result of direct rupture of the membrane by the canula together with the bad effects of the water which had found its way into the tympanic cavity. It is a well known fact that water introduced into the tympanic cavity through a traumatic rupture results practically always in suppuration of the middle ear. Such a mishap is of such importance that the writer feels justified in mentioning it.

GALVANIC NYSTAGMUS.

ACCORDING to L. William Stern (*Arch. f. Ohrenheilk.,* Band XXXIX, pages 248-284) the first one to investigate the effects of the galvanic current in producing vertigo was Ritter in 1803. The results of Ritter's investigation were published in Hufeland's *Journal f. prakt. Heilk.,* bd, XVII, 3 stueck, s. 34, under the title of "Ueber die Anwendung der Voltaischen Saule." Nearly three-fourths of a century later, E. Hitzig, 1871, published the results of his experiments in an article entitled "Ueber die biem Galvanisiren des Kopfes entstehenden Stoerungen der Muskelinnervation und der Vorsteilungen von Verhalten in Raume"—*Reichert und Dubois' Arch.,* s. 716-770. Hitzig was the first to observe that the application of galvanic electricity to the sides of the head caused vertigo, the subjective sensation of objects rolling to the side of the kathode, and that with the use of the stronger currents the subjects tended to fall towards the side of the anode. His observations were correct and have since been amply corroborated. He failed, however, to observe the associated nystagmus, which must have been present, to the side of the kathode. He explained the reaction as due to cerebral irritation; falsification of the muscle sense.

E. Hitzig, 1874, wrote a second article entitled "Untersuchungen ueber das Gehirn"—Berlin, which was a polemic against the Mach-Breuer theory; pointing out that their theory did not explain galvanic vertigo. In this he was supported by Kny, 1887 ("Untersuchungen ueber den galvanischen Schwindel"—*Arch. f. Psyschiatrie,* XVIII, s. 637-58), who claimed that galvanic vertigo was not dependent upon the semicircular canals.

J. R. Ewald, 1890, "Die Abhaengigkeit des galvanischen Schwindels vom inneren Ohr" (*Centralblatt f. d. Med. Wissenschaft,* XXVIII, s. 753-755), found that pigeons with completely extirpated inner ears did not manifest galvanic vertigo.

Both Hitzig and Ewald made careful and accurate observations. Their conclusions were correct as far as they had gone, in spite of their apparent contradictions.

The writer, in making extensive examinations with the galvanic current, has met with these same apparent contradictions and explained them in a subsequent part of this paper.

J. Pollak—"Ueber den galvanischen Schwindel bei Taubstummen" (Pflueger's *Arch.* LIV, s. 188-207, 1893)—found that normal people show upon the application of the galvanic current to the head typical head and eye movements which are not present in about 30 per cent. of deaf mutes. He concluded that the cause of the reaction must be in the vestibular apparatus.

Alexander and Kreidl, "Ueber die Beziehungen der galvanischen Reaction zur angebornen und erworbenen Taubstummheit" (*Arch f. die ges. Physiologie,* bd. 89, seite 475-492, 1902), examined deaf mutes with the galvanic current. Their method was to apply the two electrodes to the opposite sides of the head, always using the kathode to the right ear and the anode to the left. They considered only the reaction movements of the subjects and did not make any observations of the nystagmus reaction. They noted the head movement by the opening and closing current of 30 milliamperes. The reaction was considered positive when by closing the current the head moved toward the anode and when by opening the current the head moved toward the kathode.

Barany—"Phys. u. Path. des Bogengang Appar. beim Menschen," Wein, 1907, page 34-36—gives a brief outline of the improved method for the examination of the galvanic nystagmus of each ear separately and cites Neumann's experiment of galvanization of the VIIIth nerve stump after operation; however he concluded that the galvanic method of examination was of no practical value.

Neumann's experiment, cited by Barany, was made in the Politzer Clinic, 1906. He found after an operation for the extirpation of the labyrinth, that the nerve stem reacted to the negative electrode when introduced into the wound cavity, which in a measure supports the previous claim of Hitzig and Kny; that galvanic nystagmus can be produced independently of the semicircular canals. Had Neumann tried the experiment several weeks or months later, he would not have found this reaction present because of the secondary degeneration of the nerve which follows extirpation of the labyrinth. Neumann's results then would have corroborated the results of Ewald's experiments upon pigeons and those of Pollak and my own upon a certain per cent. of deaf mutes.

Alexander and MacKenzie—"Functional examination of the organ of hearing in deaf mutes," translation from the German, *Arch. of Otol.*, vol. XXXVIII, no. 6, 1908—examined deaf mutes, using the same material as was used by Pollak, Kreidl and Alexander. They found that

22 out of 51 cases showed absolutely no reaction with 12 ma.; a few of these might have shown a positive reaction with a stronger current.

About this time (1908), the writer published the results of his experiments upon a variety of cases under the title, "Klinische Studien uber die Functionspruefung des Labyrinthes mittles des Galvanischen Stromes" (*Arch. f. Ohrenheilk.*, bd. 77 and 78, 1908). The method employed by Alexander and MacKenzie was according to the method cited in this paper.

There are two methods of producing galvanic vertigo and nystagmus: (1) by the two electrodes applied to opposite sides of the head; kathode to one side and anode to the opposite side. This was the first method employed. By this method a much weaker current is required to produce a reaction than by the second method to be described. The first method will produce a positive reaction in normal cases with a current strength of from 2 to 4 ma. The reaction consists of (a) rotatory nystagmus to the side of the kathode; (b) sensation of the room rolling to the side of the kathode (pointed out first by Hitzig); (c) sensation of falling in the sagittal plane to the side of the kathode; (d) actual falling takes place to the side of the anode (pointed out first by Hitzig). This falling Barany terms reaction falling and he claims it is produced by the nystagmus; but Ewald claims it is due to hypertonus of the voluntary muscles of the side of the body corresponding to the kathode or to hypotonus of the side of the body corresponding to the anode.

By this first method the two sides are irritated simultaneously (the one by the kathode, the other by the anode) and it is impossible to tell exactly in cases of pathologic imbalance, which side is pathologically under or over irritable.

The untrustworthiness of this first method for the examination of each side separately gave rise to the second method, *i. e.*, the application of one electrode to the region of one ear and the other to some distant part, preferably away from the head. Unfortunately the wider the separation of the electrodes the more the resistance is increased and naturally the stronger the current must be applied to produce the same reaction. Barany found it necessary to use a current strength of from 10 to 20 ma. to produce a marked reaction by this second method. The writer, on the other hand, has been able to produce distinctly visible reactions with a very much weaker strength of current—from 4 to 8 ma.

The writer considers the technique of considerable importance in making the galvanic reaction, and for this reason will describe briefly the method which he employs. The method has been described before in some of his writings; however, to save the readers time in looking it up we shall review it here.

What is required?—A suitable wall plate with an accurate milliampere-meter, with the scale sufficiently large to be plainly seen. Two electrodes—one large and flat, the other small (1 cm. in diameter) and rounded, fitted into a handle with an interrupting adjustment, with cords of at least 2 meters length. The wall plate should be fitted too with a reversing switch to change the polarity.

An assistant is necessary to control the various switches of the wall plate and observe the milliampere-meter.

Good illumination, preferably from a head mirror to be worn by the observer. In using the head mirror it is necessary to have a light placed behind the subject's head. Care should be taken not to reflect the light too strongly into the eyes. I try as far as possible to direct the light slantwise on the eyeball and focus on the sclera above the cornea, so that a minimum amount of light enters the pupil.

The subject should be seated comfortably in a chair close to the wall plate and should be told that the examination is not painful; any anxiety the patient may have concerning the examination should be allayed. The operator should stand facing the subject, slightly to the left when examining the left side and slightly to the right when examining the right side. This is done in order to allow the patient to look straight ahead over the observer's shoulder, at some distant point. Abel's opaque spectacles are not needed in this examination. In order to see the sclera above the cornea it is well to have the subject's head inclined somewhat backward (15° to 20°). It might be added that the reaction is not governed in the least by the position of the head. In other words, the character and the direction of the nystagmus are the same no matter to which position the head may be put. The subject should fix his gaze upon some small object on the opposite wall.

As a preliminary, the observer should note carefully whether spontaneous nystagmus is present or not, and if present its character, direction and intensity. The observation of nystagmus should always be made with the upper lid passively elevated by the thumb of the observer, taking care not to touch the eyeball or lid edges. It must not

be forgotten that the eyeball, when too long exposed tends to become dry and this is uncomfortable to the patient. To avoid this, the writer frequently lowers the upper eyelid momentarily at regular intervals.

It is always essential to have the parts where the electrodes are to be applied thoroly moistened with warm salt water (too much cannot be used) and also the sponges of the electrodes should be kept dripping wet with the same solution. It is my practice to place the large flat electrode in the right hand of the subject to be held there thruout the examination. I do not consider it necessary to change hands when examining the opposite ear; I have never been able to note any difference whatever between using the one hand and the other or whether the right hand was used when examining the same or the opposite ear. The handle of the second electrode is held in the right hand of the examiner when examining the left ear, the small, sponge, end is placed just in front of the tragus. The examiner's left hand rests upon the frontal region of the subject's head and with the thumb is free to move the upper lid of the left eye which is gently elevated to uncover a fair portion of the sclera above the cornea. The current is started by the assistant gradually increasing the strength until a reaction is observed, then I say to the assistant "reaction present to the left, or right"—as the case may be—and he notes the reading of the milliampere meter, calling it off to me, and tells me too if the direction of the nystagmus as I find it, corresponds with the polarity used.

Up to this time I have not allowed myself to know the polarity, which is controlled by a switch in the hand of the assistant. This is done so that I may not be governed, in the least, in deciding the direction that the nystagmus should take; thereby helping to eliminate the socalled personal equation factor. Curiosity naturally prompts me to know afterwards, and I find myself looking at the instrument. This is done, too, to see that the assistant, should he be new at the work, has done his part correctly.

So we proceed examining first one ear with both the anode and kathode, then the other ear, often repeating the examination on the same individual two or three times to be absolutely certain of our result. After making this examination, occasionally coincident with it, an examination of the opening and closing nystagmus is made, using about the same strength of current or a little less than was required to cause the first reaction.

In making the galvanic nystagmus examination, I frequently in-

struct the patients to tell me of their subjective sensations, *i. e.*, to which side they saw the room rolling or to which side they felt they were falling, remembering that both of these sensations are to the side of the kathode or away from the anode and to the side corresponding to that of their nystagmus.

The reaction may manifest itself in different ways according to circumstances.

The normal individual without spontaneous nystagmus, except in the extreme lateral position of the eyeballs, should show when looking straight ahead:

(a) Rotatory rythmic nystagmus to the same side when the kathode is used with a current strength of from 4 to 8 ma.

(b) Rotatory rythmic nystagmus to the opposite side when the anode is used, and with a current strength equal to that required to produce the same degree of nystagmus to the same side when the kathode is used.

Normal people should show a perfect balance of reactions to the two sides and an equal balance between the kathode and anode; for instance, if 4 ma. kathode to the left side produces rotatory nystagmus to the left side, then 4 ma. anode to the left side should produce the same degree of nystagmus to the (opposite) right side and 4 ma. kathode to the right side should produce the same degree of nystagmus to the right side. This is true of course only when the technique is perfect. Carelessness in keeping the parts equally wet with salt solution or applying the electrodes to different places on the two sides (*i. e.*, a favorable location on one side and an unfavorable location on the other) or malobservation of the eye movements during the examination may lead to failure to recognize the accurate balance noted above. However, experience and earnestness should soon teach one the importance of carefulness.

That normal people show no spontaneous nystagmus when looking straight ahead and that they should manifest nystagmus to the opposite side when the anode is used proves that there must be normally a definite amount of bilateral tension or tonus on the two sides. This is located in the nerves and, in a measure, their terminals in their end-organ (special sense epithelia in the ampullæ of the semicircular canals). This same tonus is present in all nerves thruout the organism. It may vary somewhat in degree in different individuals but is ever present in every healthy individual. In the case of the vestibular

nerve and its endorgan this tonus is capable of accurate measurement and is found to be somewhere between 4 and 8 ma.; which is the intensity of current required to upset the balance between the two opposite or antagonistic nerves.

In pathologic cases we find a variety of deviations from the normal reactions. The writer, after making careful observations with the galvanic current upon a great many normal and pathologic cases, was able by this method of examination to divide the pathologic cases into three primary groups, namely:

I. Those with a pathologically over-irritable inner ear of one side.

II. Those with a pathologically under-irritable inner ear (destroyed inner ear) of one side but with the nerve still irritable. The case examined by Neumann, where after extirpation of the labyrinth he found the nerve stem still reactive to the cathode, comes under this group.

III. Those with negative irritability of both inner ear and nerve. The pigeons examined by Ewald after total extirpation of the semicircular canals fall under this group.

This same classification of pathologic conditions is possible in a measure also with the turning method of examination. Details of the individual cases examined by the writer may be found in a former paper by the writer ("Klinischen Studien ueber die Functionspruefung des Labyrinthes mittles des galvanischen Stromes"—*Archiv f. Ohrenheilk.*, Bd. 78, 1909).

There are other secondary groups, including the cases of double sided affections, etc., but to cite them all would only tend to confuse the beginner.

Let us represent the normal reactions graphically, as follows:

R. E.	L. E.
*K. 4 ma. Rot. R.	K. 4 ma. Rot. L.
A. 4 ma. Rot. L.	A. 4 ma. Rot. R.

*The numeral 4 is here used to show the strength of current because it is approximately normal and can be used to better advantage to show what is intended than the numeral 5 or some other odd figure. We could use quite as well the numeral 6.

R. E. = right ear, L. E. = left ear, K. = kathode, A. = anode, Ma. = milliampere, Rot. L. = rotatory nystagmus to the patient's left, Rot. R. = rotatory nystagmus to the patient's right.

If it takes 4 ma. with the anode to the right ear to suppress the normal tonus sufficiently to permit the normal tonus of the left side to produce a rotatory nystagmus to the left, we may conclude that the strength of the normal tonus to the right side is equal to 4 ma. On the other hand, if 4 ma. with the kathode to the right ear is required to produce rotatory nystagmus to the right side, we may conclude that 4 ma. kathodal irritation to the right side plus the normal tonus of the right side is sufficient to overbalance the normal tonus of the left side. In either of the above instances it is readily seen that a difference of 4 ma. tension, whether it be taken from or added to the normal, will result in a sufficient overbalance to produce rotatory nystagmus to the side of the increase of tension or to the side opposite the diminution.

I. A pathologic case with the history of right-sided middle ear suppuration and attacks of vertigo may present the following reactions:—

R. E.	L. E.
K. 2 ma. Rot. R.	K. 6 ma. Rot. L.
A. 6 ma. Rot. L.	A. 2 ma. Rot. R.

This case shows first a difference between the kathodal reactions of the two sides of $6 - 2 = 4$ ma., which is just about enough to produce an overbalance and cause the subject to manifest a very slight spontaneous nystagmus to the right side; secondly, a difference between the kathodal and anodal reactions of the same side of $6 - 2 = 4$ ma.; thirdly, the kathodal reaction of one side just balances with the anodal reaction of the opposite side, which is just what we find in this group of cases.

By comparing the figures in the second case with those in the first (normal) we can estimate the amount of pathologic irritability.

If it takes normally 4 ma. with the kathode to the right ear to produce an overbalance of impulses to the right side, then it follows, in pathologic case I, where but 2 ma. is required, that the difference between 4 ma. and 2 ma. or 2 ma. represents the amount of pathologic irritation of the right side. Furthermore, if it takes 4 ma. with the anode to the right side to suppress the normal tonus, then it follows in this same case where 6 ma. is required that the 2 additional ma. must be used to overcome that amount of pathologic irritation of the right side. In the patient just examined we may conclude that the amount of pathologic irritation of the right inner ear is equal to 2 ma. and represent the reactions algebraically as follows:

Right ear:

*R.N.T. 4 ma. + R.K.T. 2 ma. + R.P.T. 2 ma. = R.T.T. 8 ma.

R.T.T. 8 ma. — L.N.T. 4 ma. = R.B.T. 4 ma. or surplus of 4 ma. tension to right side, which is sufficient to cause a rotatory nystagmus to the right side.

R.N.T. 4 ma. + R.P.T. 2 ma = R.T.T. 6 ma.

R.T.T. 6 ma. — R.A.T. 6 ma. + L.N.T. 4 ma. = L.B.T. 4 ma.

Since the 6 ma. with anode to the right side just neutralizes the + 6 ma. total tension on right side, there is left 4 ma. of normal tension on the left side to produce rotatory nystagmus to the left.

Left ear:

R.N.T. 4 ma. + R.P.T. 2 ma. = R.T.T. 6 ma.

L.N.T. 4 ma. + L.K.T. 6 ma. = L.T.T. 10 ma.

L.T.T. 10 ma. — R.T.T. 6 ma. = L.B.T. 4 ma.

The left total tension exceeds the right total tension by 4 ma., which is sufficient to cause rotatory nystagmus to the left side.

R.N.T. 4 ma. + R.P.T. 2 ma. = R.T.T. 6 ma.

L.N.T. 4 ma. — L.A.T. 2 ma. = L.T.T. 2 ma.

R.T.T. 6 ma. — L.T.T. 2 ma. = R. B. T. 4 ma.

which is sufficient to cause a reaction to the right side.

II. A second case, with the history of chronic middle ear suppuration of the right ear combined with complete deafness of the same ear and vertigo of recent date, associated with spontaneous rotatory nystagmus to the left side, may show the following reactions:

R. E.	L. E.
K. 7 ma. Rot. R.	K. 1 ma. Rot. L.
A. 1 ma. Rot. L.	A. 7 ma. Rot. R.

Comparing these figures with the normal we find:

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*R.N.T. = normal tension of right side.

R.K.T. = kathodal tension to right side.

R.P.T. = pathologic tension of right side.

R.T.T. = total tension of right side.

L.N.T. = normal tension of left side.

R.B.T. = balance of tension to right side, or right side irritation exceeds that of left side by 4 ma.

R.A.T. = anodal irritation, or minus tension of right side.

L.K.T. = kathodal irritation or tension to left side.

L.T.T. = total tension to left side.

L.A.T. = anodal irritation or minus tension to left side.

L.B.T. = balance of tension to left side.

(1), with the kathode to the right ear, that to obtain a reaction* a much stronger current is required; in fact, 7 ma. — 4 ma. = 3 ma., more than normal.

(2), with the anode a proportionately weaker current is required (4 ma. — 1 ma. = 3 ma.).

(3), with the kathode to the left ear but 1 ma. current strength is required to produce a reaction (increase the nystagmus to the left), which is 3 ma. less than is required normally.

(4), with the anode 3 ma. more than normal (4 ma.) is required.

From these reactions it is evident that though the irritability of the right side is diminished, it is not entirely lost. Furthermore, that since the right inner ear has been destroyed (thru labyrinth suppuration) this remaining irritability must lie elsewhere than in the inner ear, no doubt in the nerve.

Comparing the reactions of the left side with those of the right, we find the same reactions with the same figures but reversed polarity or with the same polarity but with reversed figures. In other words, 7 ma. kathode to the right side will produce the same reaction as 7 ma. anode to the left. Again, 1 ma. anode to the right side, will produce the same character and amount of reaction as 1 ma. kathode to the left ear. Again, if the kathodal reaction is 1 ma. and the anodal is 7 ma. on one side, the reaction on the opposite side would be kathodal 7 ma. and anodal 1 ma.

The explanation of reactions may be found in the following equations. Representing the amount of pathologic destruction in the right inner ear by 3 ma.

Right ear:

R.N.T.4 ma. — †R.P.L.T.3 ma. +R.K.T.7 ma. = R.T.T.8 ma.
R.T.T.8 ma. — L.N.T.4 ma. = R.B.T.4 ma.
which is just sufficient to cause a rotatory nystagmus to the right.

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*A reaction in this class would be indicated by a cessation of the spontaneous nystagmus to the left or by a reversal of the direction of the nystagmus.

A reaction in the case of anode to the right ear would be indicated by a perceptible increase in the nystagmus to the left.

It must not be forgotten that all of my observations have been made with the patient looking straight ahead and not to the sides as has been done by many of the other authors.

†R.P.L.T. = right pathologic loss of tonus or tension (loss of tonus from destruction of inner ear).

R.N.T.4 ma. — R.P.L.T.3 ma. — R.A.T.* 1 ma. = R.T.T.0 ma.
L.N.T.4 ma. — R.T.T.0 ma. = L.B.T.4 ma.
which necessarily produces spontaneous nystagmus to the left.

Left ear:
R.N.T.4 ma. — R.P.L.T.3 ma. =R.T.T.1 ma.
L.N.T.4 ma. + L.K.T.1 ma. = L.T.T.5 ma.
L.T.T.5 ma. — R.T.T.1 ma. = L.B.T.4 ma.
which must produce a nystagmus to the left.
R.N.T. 4 ma. — R. P.L.T. 3 ma. = R.T.T. 1 ma.
L.N.T. 4. ma. — L.A.T. 7 ma. = L. T.T. — 3 ma.
R.T.T.1 ma. — L.T.T.— 3 ma. = R.B.T.4 ma.
which difference in tension is just sufficient to cause rotatory nystagmus to the right side.

III., a third case presenting the same history as the last but of much longer duration (where the attack of vertigo has probably antedated the examination two or three months) will show the following reaction:

R. E.	L. E.
K. 12 ma. or more, no reaction.	†K. 2 or 3 ma. Rot. L.
A. 12 ma. or more, no reaction.	‡A. 12 ma. or more, no reaction.

Analysis of this reaction shows that the right inner ear and nerve cannot be made to react to a current strength of 12 Ma. and more from which it may be safely concluded that the vestibular nerve has undergone complete secondary degeneration. The normal tonus is still present in the left inner ear and nerve and it can be made to react to both the kathode and the anode; however, by the most complete anelectrotonus of the left nerve a reaction to the right cannot be produced.

In examining deaf mutes I found quite a number who did not react to 20 ma. and even 30 ma. Besides, these were not all cases due exclusively to early meningitis; for the great majority in whom no reaction was present were due to other causes.

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*R.A.T. = anodal tension, is always a negative one and must be represented with a minus sign in front of it.

†A barely perceptible spontaneous nystagmus to the left of short excursions and wide intervals, is increased to that of longer excursions and closer intervals.

‡With the anode the strongest currents cannot reverse the direction of the nystagmus, however they may cause a cessation of the very slight spontaneous nystagmus which is present and from this standpoint it might be said that a reaction is possible with a current strength of somewhat less than 12 Ma.

In a case where the one or the other side cannot be made to react, with either the kathode or anode, using the strongest currents we may safely conclude that it is one of primary or secondary destruction of the vestibular nerve.

OPENING AND CLOSING NYSTAGMUS.

The examination of the opening and closing nystagmus is but one step further in the examination than that which has already been cited. The same apparatus is used as in the previous method of examination. Ascertain by the previous method the least current strength required to produce a positive reaction where a positive reaction is possible.

For instance, in a normal case with the patient reacting to 4 or 6 ma., the reactions should balance and may be recorded as follows:

R. E. With 4 to 6 ma.	L. E.
*K.C.N. = K.O.N.	K.C.N. = K.O.N.
A.C.N. = A.O.N.	A.C.N. = A.O.N

The explanation of this reaction is as fol'ows: Kathodal closing nystagmus is the nystagmus which is caused by closing the current with the kathode applied to the ear; it is rotatory in character and is directed towards the side of the kathode. With the use of the stronger currents the nystagmus is associated with the sensation of falling in the sagittal plane towards the kathode, while actual (reaction) falling occurs in the opposite direction, away from the kathode. This latter fact was pointed out long since by numerous writers, including Kreidl and Alexander.

Kathodal opening nystagmus is the nystagmus which occurs for a few seconds after the breaking of the current. It is most intense at the moment the current is interrupted. It is rotatory and is directed towards the opposite side (away from the kathode). With the stronger currents it is associated with the sensation of falling in the sagittal plane away from the kathode, while actual falling occurs towards the kathode.

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*K C.N. = kathode closing nystagmus.
K.O.N. = kathode opening nystagmus.
A.C.N. = anode closing nystagmus.
A O.N. = anode opening nystagmus.
$>$ = greater than.
$<$ = less than.

Anodal closing nystagmus is the nystagmus which is produced by closing the current with the anode applied to the ear. It is rotatory in character and is directed towards the opposite side; with the stronger currents it is associated with the sensation of falling in the sagittal plane towards the opposite side, while actual falling occurs towards the same side.

Anodal opening nystagmus is the nystagmus which results from opening or breaking the current with the anode applied to the ears and lasts for a few seconds but is most intense at the instant of breaking the current. It is rotatory in character and is directed towards the same side. With the stronger currents it is associated with the sensation of falling in the sagittal plane towards the same side while the actual falling occurs towards the opposite side (away from the anode).

In the first type of pathologic case recorded, that is with a pathologically over-irritable right inner ear, we find the reaction to be:

R. E. With 4 to 6 ma.	L. E.
K.C.N. > K.O.N.	K.C.N. < K.O.N.
A.C.N. < A.O.N.	A.C.N. > A.O.N.

In the second type of case suffering with pathologically destroyed right inner ear but with intact nerve, we find the reaction to be:

R. E. With 4 to 6 ma.	L. E.
K.C.N. < K.O.N.	K.C.N. > K.O.N.
A.C.N. > A.O.N.	A.C.N. < A.O.N.

In the third type of case suffering with pathologically destroyed right inner ear combined with secondary degeneration of the vestibular nerve, we find the reaction to be:

With current strength of 12 ma. or more.

R. E.	L. E.
K.C.N. and K.O.N. not present.	K.C.N. > K.O.N.
A.C.N. and A.O.N. not present.	A.C.N. < A.O.N.

Strictly speaking there is no K.O.N. or A.C.N. upon the left side in this group of cases. The most that may be expected by opening the current with the kathode or closing it with the anode is the cessation of the existing nystagmus to the left side, which we may accept as a positive galvanic reaction. The production of a nystagmus to the right side, however, in the case of destroyed vestibular nerve of the right side is impossible by any method of irritation of the destroyed nerve or inhibition of the opposite nerve.

UTRICULUS AND SACCULUS.

THE utriculus and sacculus are the two membranous sacs located within that part of the osseous labyrinth known as the vestibule. The utriculus is located just anteriorly to the semicircular canals and is in direct communication with them through five openings. The sacculus is located anteriorly to the utriculus and posteriorly to the ductus cochlearis and is in direct communication with the latter through the ductus reuniens. The utriculus and sacculus are in communication with each other through the ductus utriculo-saccularis.

The utriculus and sacculus each contain an endorgan known as a macula acustica, composed of a group of ciliated neuro-epithelia with alternating supporting cells. The cilia of the neuro-epithelia are short.

The maculæ are somewhat elliptical in shape with the long axes running at right angles to each other. On the surface of each macula is a jelly-like mass in which is suspended small calcium carbonate crystals (otoliths or statoliths).

These sacs are phylogenetically the oldest part of the ear labyrinths; the simplest form of which is that found in the Ctenophores (jelly fish) for which Verworm, because of its function, suggested the name statocyst. That these sacs perform a function distinctly separate from that of the semicircular canals has been shown by the researches of Delage, Verworm, Breuer, Mack, Engelman, Kreidl, Lee, Kubo and others.

As early as 1874 Mack[1] expressed the view that the sensation of angle accelerations (turnings) is mediated probably thru the three semicircular canals and that of progressive movements presumptively thru the sacculus. "Die Empfindungen der drei Winkelbeschleunigungen werder wahrscheinlich durch die Ampullennerven der drei Bogengaenge die Empfindungen der Progressivbewegungen muthmasslich durch den Sacculus vermittelt."

Breuer[2] simultaneously expressed the same view and, too, he believed that reflex contrarolling movements of the eyes, "reflectorischen Stellungsveraenderungen der bulbi," (observed also by Graefe[3], Skrebitzky[4] and Nagel[5]) which are manifested by changes of position of the head are produced reflexly from the ear labyrinth—macula

acustica and the otolithic mass. To Breuer, these reflex contrarolling movements of the eyes were an argument in favor of the supposed function of the macula acustica and otolithic mass as expressed by himself and Mach. We shall refer to Breuer again later.

Carl Chun[6], in 1880, experimented upon the ctenophores in the Gulf of Naples; he held the opinion that the otocyst in the invertebrates probably performed an equilibrium function.

(Henry Sewall[7] experimented upon the ears of fishes (young sharks and skates) at the marine laboratory of the Johns Hopkins University at Beaufort, N. C., in 1881, and on the Chesapeake Bay in 1883.) He claimed theories essentially similar to Goltz, Crum-Brown and Breuer, that the endolymph within the membranous labyrinth presses with every new movement of the head with greater or less force upon the auditory nerve endings within definite ampullæ of the canals and thus mechanically stimulates them. (He also attempted the removal of the otoliths from the ~~vestibular sacs~~ ears. In some cases he was able to produce equilibrium disturbances, while in others he failed.) Although his experiments were ingenious, on the whole they led him rather to uncertainty than to any definite conclusions.

W. James[8], of Harvard University, in 1887, found that of 200 normal people who were subjected to turning but one only was free of vertigo, whereas of 517 deaf mutes subjected to the same turning, 186 had absolutely no vertigo. He learned further from many of these (186), that when they were under water they suffered an indescribable confusion and anxiety which was relieved only when the head came above the surface. Similar experiments subsequently conducted by others, and particularly Breuer, corroborated the observations of James. Nothing could be more conclusive to show that the ear contains an organ for orientation in space which, when lost, materially handicaps the subject. Furthermore that the eyes are capable in a measure of overcoming the loss of equilibrium which results from a complete destruction of the ears.

Yves Delage[9] in 1886 conducted a series of interesting experiments upon cephalopods and crustaceans. Among other experiments he removed the otocysts from a crab and found that the animal was but slightly disturbed during the act of crawling, but during attempts at swimming the crab made all sorts of irregular movements. He then blindfolded the animal and found the disturbances still more pro-

nounced; whereas normal crabs blindfolded made the same regular movements but somewhat slower and more cautiously than when the eyes were not blindfolded. He repeated these experiments on other forms of crustaceans. The results of his experiments proved to him that the otocyst is an organ for orientation in space while the eyes and the antennæ (feelers) are capable of correcting, in a measure, for such loss of orientation which results from the loss of the otocysts.

The work of Delage prompted T. H. Engelmann[10] to publish an article wherein he cites his own observations made several years before, upon the otoliths of the ctenophores. Engelmann held that the otolith and the sac which holds it perform a function of body equilibrium—"den allgemein als Otolithen bezeichneten, am aboralen Pol des Ctenophorenkoerpers gelegenen Kalkkoerper fuer einen die Ert haltung des Koerpergleichgewichts vermittelunden Apparat."

Max Verworm's[11] experiments upon the ctenophores (euchris multicornis, bolina hydatina, beroea ovata) led him to conclude that the otolith and otocyst performed the function of equilibrium in these animals, endorsing the prior supposition of Engelmann. Because of their function he suggested the terms statolith and statocysts as more appropriate than otolith and otocyst. He experimented further with these animals to determine if possible whether these organs performed in addition an acoustic function. He failed, as did also Chun, to produce any response to the loudest tones and noises.

Jacques Loeb[12] experimented upon sharks (scyllium canicula and catalus) in the winter of 1890-'91 at the Zoölogical Station at Naples, resecting certain parts of the brain (the cerebral hemispheres, the middle brain, the medulla oblongata in the region of the eighth nerve and the eighth nerve). He found as the result of section of the eighth nerve, forced movements of the eyes to the destroyed side and the members (fins) to the opposite side, together with rolling movements and circus movements (around a ring) towards the operated side during attempts of the animals to make voluntary movements.

Loeb in a separate article (Geotropismus bie Thieren) recites his experiments upon the inner ears of the sharks. When the otoliths of one side were removed, he found the animals deviated laterally downward to the operated side from 20° to 50°. When the otoliths of both sides were removed the animals failed to retain the normal horizontal position of equilibrium even when they were so placed. These operated

animals furthermore failed to show the normal forced (reflex) movements of the eyes (contrarolling movements) manifested by changes of position.

Kreidl[13] likewise observed this lack of balance in sharks with double side loss of otoliths; he found that the sharks would as frequently swim or lie on their backs as they would take any other position.

F. S. Lee[14] in 1893 conducted experiments mostly upon dog sharks galens canis) and some five specimens of skates (raja erinacea). In his introduction he claims that "a complete theory of the socalled 'equilibrium' function of the internal ear ought to be competent to explain three classes of facts, viz., the equilibrium phenomena of the resting body, those of the body moving in a straight line, and those of the body moving in a curve. The first comprises the statical, the last two dynamical phenomena."

He points out the fact that birds and fishes are naturally best adapted for study because these animals spend so much of their time in a fluid medium out of contact with a solid supporting surface.

Lee noted compensatory eye and fin movements. He endorses Breuer's work and conclusions and sums up under the heading, "Equilibrium Function of the Ear."

"II. STATIC FUNCTIONS.

"(a) *Normal:* Sensations of position of the body in space accompanying corresponding positions of the eyes and fins; varying according to nerve stimulated (?).

"(b) *Pathological:* Abnormal sensation causing lack of orientation and abnormal position of the eyes, fins and trunk; varying according to the nerve thrown out of function. (Localization of statical function in otolithic parts; stimulation continual.)

Breuer[15] in 1891 in an interesting article on the subject of the functions of the otolithic apparatus, reviews the literature up to that time, and sets forth his own theories as to the subject. Quoting as accurately as I can from his resumé:

"RESUME, PAGE 268.

"1. There exists positively specific sensations for the position of the head to the vertical and for progressive (straight line) movements.

"2. The topographic arrangement of the otolithic apparatus makes

it most probable that they, like the semicircular canals, have to do with the perception of local or spacial conditions. Their structure makes it probable that the gravitation of the otoliths is the producing irritant.

"3. The Ausfallserscheinungen (*i. e.*, the phenomena resulting from the loss of function) in frogs and birds with destroyed labyrinths and of deaf mutes, proves that the labyrinth mediates the perception of position in space, which in the cases cited (deaf mutes) were completely absent, when by immersing under water they who were formerly orientated lost for the most part their sensations of gravitation.

"4. Every position of the head corresponds in man to a definite combination of gravitations upon the four maculæ. When we accept that gravitation of the otolithic plates drags on the cilia and excites nerve endings, producing an irritation which is conveyed to the center for the conception of position, then it appears that the sacs of the labyrinth are (collectively) an organ entirely adapted as one of special sense for the perception of our location in space."

I might go on page after page reviewing the experiments and publications of Kreidl[13], Crum-Brown[16], Bunting[17], Bethe[18], Rawitz[19], Lautenback[20], Alexander and Kreidl[21], Cyon[22], Panse[23], Ach[24], Zoth[25], Froelich[26], and others, but instead I will proceed directly to a brief discussion of what may be considered the most important and conclusive work accomplished in this direction (the physiology of the vestibular sacs). I refer to that done by Ino Kubo[37].

The experimental research work of Kubo is the most exact and thoro of its kind. He spent the winter of 1905 and 1906 at the K. K. Zoölogical Station in Triest experimenting on living fish (scyllium canicula, acanthias vulgaris, mustelus laevis, rhombus maximus, pleuronoctes platessa, raja clarata, torpedo marmorata and petromyzon marinus).

1. His experiments consisted of studying the eye movements during active and passive turnings of the animals.

2. Experiments upon the semicircular canals and the ampullæ by
   (a) Thermic irritation.
   (b) Mechanical irritation.
   (c) Galvanic irritation.

3. He studied the deviations of the eyes when the animals were passively turned to different positions and made experiments on the otoliths of the vestibular sacs (utriculus, sacculus and legena).

It is that part of Kubo's research which dealt with a study of deviations of the eyes and experiments upon the sacs which interests us more especially at present.

The exactness of Kubo's methods and the logic of his conclusions practically preclude any adverse discussion; I shall therefore briefly outline his work. For various reasons some of the fish were better adapted for one form of study than others and vice versa; so that I shall recite the results obtained generally rather than those obtained in each individual fish.

EYE POSITIONS (DEVIATIONS).

When the fish was held in the position of long axis horizontally and with the belly down, the eyes assumed their normal position.

When the fish was turned to the vertical position, *head up,* the eyes rolled downward and remained there as long as the fish was held in that position.

When the fish was turned to the vertical position, *head down,* the eyes rolled upward and remained there as long as the fish was held in that position.

When the fish was turned to the horizontal position with the belly up the eyes resumed the primary position as in the case of belly down.

In the side position (with long axis horizontal) the eyes deviated in the vertical axis, so that the upper eye deviated downward and the under eye upward.

To show that these deviations of the eyes were not dependent upon the semicircular canals Kubo then removed the membranous canals together with their ampullæ, when he found that the identical deviations of the eyes occurred as in the first instance. He next removed the sacs of both sides, when he found the deviations ceased to occur.

When the sacs of one side only were removed the deviation occurred, but not so pronounced as when both sides were intact.

KUBO'S EXPERIMENTS ON OTOLITHS.

1. He exposed the membranous sacs to full view and observed the gliding movements of the otoliths.

When changing the position of the fish from the horizontal (belly down) to the vertical, *head up,* he was able to see plainly a gliding movement of the otoliths of the sacculus downward. In the side positions the movement of the otoliths in the sacculus was slight but visible.

Briefly, he found the otolithic mass to glide or gravitate in a direction corresponding to the movement of the eyes or contrary to the movement of the head.

2. *Direct manipulation of the otoliths with small cotton protected probes.*

When the otolithic mass was pushed with the probe in definite directions the eyes deviated in the corresponding directions. For instance, when the otoliths of utriculus (fish in the horizontal position, belly down) were gently shoved forward the eyes would deviate upward, the position corresponding to that which occurs when the head is turned downward and the otolith mass of the utriculus naturally gravitates forward.

Again, when otoliths of the sacculus were gently shoved backward the eyes deviated downward; the position corresponding to that which occurs when the head is turned upward.

Briefly, every artificial movement of the otoliths produced reflex deviations of the eyes corresponding to those produced by the same natural movements of the otoliths which follow changes in position of the head.

3. *Experiments after partial or total extirpation of the otoliths.*

After the removal of the otoliths of the sacculus the rotation of the eyes by the position *head up* was very indistinct, while the rotation of the eyes by the position *head down* was quite distinct; from which he concluded that the rolling of the eyes in the position of the head down was due principally to the forward gliding of the otoliths in the utriculus.

After the removal of the otoliths of the utriculus from one side the deviations of the eyes by the position head up and head down was very indistinct. After the removal of all otoliths from one side the deviations of the eyes by the position of the head up and head down occurs but slightly on the unoperated side only. In the side positions also only on the unoperated side.

By turning the fish in the horizontal plane after total extirpation of the otoliths of one side there occurs no deviation bulbi horizontalis by turning towards the operated side, while turning toward the healthy side produces distinct deviation bulbi bilateralis and in this case the deviation is stronger in the eye corresponding to the unoperated side.

4. *Electrical irritation of the nerve branches after the removal of the otoliths together with the ampullæ.*

As previously observed, one sees two groups of nerve fibres in the floor of the utriculus and one group in the sacculus. From the anterior group branches go to the macula utricularis and to the two ampullæ (anterior and horizontal) and from the posterior group branches go to the macula saccularis and the ampullæ of the posterior canal. Kubo observes that one cannot irritate the individual nerve fibers of each group; however, he was able to, at least, irritate separately the three groups of fibers, the results of which follow:

1. By electrical irritation of the nerve fiber group to the utriculus of one side, the nasal pole of both eyes turned upward corresponding to the position of the head down.

By electrical irritation of group III (the group of fibers to the utriculus) the eyes of both sides deviated horizontally to the side opposite the irritation, corresponding to that which appears by turning of the head to the operated side.

By electrical irritation of group III (the group of fibers of the sacculus) the nasal pole of both eyes deviated downward, corresponding to the position of head up.

When the contents of the vestibulum were curetted entirely away and the remaining nerve stem was electrically irritated, the eyes rolled in the horizontal plane as in the case of rabbits and pigeons.

CONCLUSION.

Among other things Kubo observed that "also with fish every body position corresponds to an exact eye position and when the otoliths are removed or the entire vestibular apparatus is destroyed this relationship is lost." He then cites a list of authors who had since the time of Hunter[27] observed and studied these eye movements (contrarolling) which occur as the result of inclinations of the head. He gives credit to Mack and Breuer as the first authors to point out separate functions for the semicircular canals and for the otolithic apparatus. "The single direct *proof* until now (of the functions of the otolithic apparatus) was Kreidl's iron filing experiments upon crabs."

"Die Verschiebung oder Gleitung der Otoliten ist wenigstens bei Rochen und Acanthias vulgaris bei Lagewechsel des Korpers sicher zu konstatieren; und die kuensiliche Verschiebung der Otoliten hat dieselbe Bulbusdrehung zur Folge wie der Lagewechsel des Koerpers —wenigstens fuer die Lagen 'Kopf oben und Kopf unten.' Das ist

ein neuerlicher Beweis dass die Gleitung der Otoliten die Nervenendigungen reizt, wie dies Breuer annimmt. Aus den Versuchsresultaten geht hervor, dass der Zug odor die Spannung der Haarzellen durch die Gleitung der Otoliten als normaler Reiz zu betrachten ist. Je nach der Gleitrichtung muessen die verschiedenen Nervenendzweige gereizt werden, deren reflektorische Funktionen (oder Verbindungen) ungefaehr durch elektrische Reizung zu bestimmen sind; Z. B. in der 'Lage Kopf unten' muss der Otolit des Utriculus nach vorn gleiten und diejenigen Nervenzweige reizen, die durch elektrische Reizung in der Bauchlage die Bulbi so zu drehen veranlassen, dass der nasale Bulbuspol nach oben geht ('I. Gruppe'); diese Nervenzweige treten zur Macula utriculi in Beziehung. In der Lage 'Kopf oben' gleiten die beiden Otoliten des Sacculus und Utriculus kaudalwarts, allein der Otolit des Sacculus uebt den wirksamen Zug auf die Haarzellen der Macula sacculi aus, wie die kuenstliche Verschiebung des sacculaeren Otolits und die elektrische Reizung der Nervenzweige im Sacculus ('III. Gruppe') uebereinstimmend gezeigt haben. Fuer die Deviatio bulbi bilateralis horizontalis kommt die II. Gruppe in Betracht; es ist jedoch schwierig, in diesem Falle die horizontale Ampulle auszuschalten, die selbst von der II. Gruppe die Nervenendzweige aufnimmt. Die Bulbusstellungen in den Seitenlagen kœnnten von der kombinierten Wirkung der beiden Otoliten (des Utriculus und Sacculus) herruehren.

Die Macula lagenae entzieht sich bei Fischen dem Experiment; bei der von Breuer festgestellten Tatsache, dass die drei Otoliten in den drei Dimensionen des Raumes orientirt sind, wird man anzunehmen das Recht haben, dass diese Apparate zur Wahrnemung der Kopf— bzw. Koerperlage dienen. Die Reizung der maculae durch die Gleitung der Otoliten ruft reflekttorisch die eigentuemlichen Bulbusstellungen hervor.

Wie Lee beschreibt, ist die Deviatio bulbi bilateralis horizontalis beim Drehen des Tierkoerpers in der Horizontalebene vergaenglich temporaer aber man hat kein Recht, deswegen nur dem horizontalen Bogengang (resp. seiner Ampulle) eine besondere dynamische Funktion (d.h. die Wahrnehmung der Kreisbewegung) zuzuschreiben; denn meine Versuche haben gezeigt, dass bei Drehung des Tierkoerpers in der Seitenlage oder in der lage 'Kopf oben' oder 'unten' eben falls Deviationen der Bulbi von verganglichen Natur auftreten. Man wird annehmen muessen, dass beim Drehen die Bogengaenge

(mit ihren Ampullen) mit dem Otolitenapparate stets gemeinsam in Taetigkeit versetzt werden, wobei die Bogengaenge fuer die Wahrnehmung der Winkelbeschleunigung und die Otolitenapparate fuer die Wahrnehmung der Lage (wahrscheinlich auch der geradlinigen Bewegung) bestimmt sind.

RESUME.

Paragraphs 1, 2, 3, 5 and 6 concern more the subject of eye movements associated with the physiology of the semicircular canals; the remaining paragraphs given below pertain more to that of the sacs.

4. The positions of the eyes are definite for every body position, as in the case of rabbits.

7. The maximal deviation of the bulbus (especially in the side position) goes back considerably after a short time.

8. After the extirpation of the semicircular canals there still occurs these same altered eye positions (contrarolling) always according to the definite body positions. They diminish perceptibly after the removal of the otoliths of one side and disappear altogether after the complete removal of the otoliths of both sides.

9. The gliding movements of the otoliths are actually visible by change of position of the body in Rochen and Acanthias. Gliding of the otoliths artificially produced (while the animal is in the normal horizontal position with belly down) produces the same eye movements which occur from the natural gliding movements of the otoliths.

10. Each group of nerve endings in the vestibulum reacts, after the removal of the otoliths, to electric irritation with a definite eye movement.

11. The alterations of eye position with changes of body position are produced reflexly by a gliding movement of the otoliths (at least) from the macula utriculi and sacculi.

Clinical observations of contrarolling movements of the eyes in man have been made by Barany[28]. He had a specially designed instrument constructed by Schwartz, of Vienna, for the accurate measurement of the normal contrarolling movements, which had been observed by many others (see above), and that these are perceptibly diminished in cases of one sided labyrinth destruction, however he has not written much upon the subject. It is to be hoped that he will sooner or later give us an extensive report of his investigations along this line.

The writer[29] examined clinically the following character of cases—

(1) Those with one sided labyrinth destruction, (2) those with double sided labyrinth destruction (selected deaf mutes), and (3) those with labyrinth diseases showing an increased reactibility of the vestibular apparatus—for the purpose of ascertaining to what degree equilibrium is dependent upon the semicircular canals and the vestibular sacs. Similar efforts had been previously attempted by Frey and Hammerschlag[30], Krotoscheiner[31], Kummels[32], Von Stein[33], Pollack[34], Kreidl[35], Alexander and MacKenzie[36], and others whose experiments had been made principally upon deaf mutes. The writer's own clinical examinations differ in some particulars from those attempted by previous investigators.

First of all patients showing the least evidence of hysteria, neurasthenia, syphilitic cerebral or spinal affections, visual defects, natural clumsiness or poor intelligence were excluded from the list. (2) To ascertain these facts the history of the patient was most carefully taken, the vision determined, and in some cases the eyegrounds examined. The pupillary and deep reflexes were examined as also coördination.

When we consider that equilibrium may be disturbed by a lesion of any one of the three peripheral sense organs of orientation (eyes, vestibular apparatus, kinesthetic sense organs, in the deep muscles, joints, etc.) or their centripetal tracts to their terminals in the brain, we appreciate how important it is, in making these clinical investigations, to include an examination of vision, pupillary and tendon reflexes.

Furthermore, since the examinations to be made were rather exhaustive, it was necessary to select intelligent patients, free from any acute ailments that might in any way tend to hasten fatigue.

Again, it was necessary to examine the patients with one sided labyrinth destruction at a period sufficiently remote from the acute affection to avoid confusing the acute equilibrium disturbances with those which I have termed the late.

The condition of the labyrinth was determined by the usual functional examination methods as practiced in Professor Alexander's clinic. I was doubly certain of most of the cases of one sided labyrinth destruction from the fact that they had previously undergone the operation for its removal.

The equilibrium was determined by the following tests: standing with feet together and eyes closed (Rhomberg), standing upon one foot then the other, gait forward, gait backward, hopping upon one foot forward and backward, all of these tests were made both with open

and closed eyes. Elevation upon the Alexander-Stein goniometer with face toward the instrument, first with eyes open then with eyes closed, with face away from the instrument, with right side toward the instrument and with left side toward the instrument; all of which were made with eyes open and with eyes closed. To avoid slipping on the goniometer the patients were examined with bare feet, and the instrument was dusted with rosin. In some cases the examinations were repeated in order to be absolutely certain of the results. Many of the patients were examined repeatedly at wide intervals. The time required in collecting and examining the material extended over a period of ten months.

Without going into too many details, individuals with a one sided labyrinth destruction showed diminished ability to equilibrate, while those with double sided labyrinth destruction showed marked disturbance, in some cases amounting to an almost complete loss of equilibrium when the eyes were closed. These disturbances were always more marked when the eyes were closed than when they were open.

The equilibrium tests enumerated did not include rotational movements, so we must exclude the semicircular canals as a factor in these cases. No doubt these patients would have shown the same degree of equilibrium disturbances by rotational movement tests (determined previously by James and Breuer and others) as by the tests selected.

Since the patients showed positive equilibrium disturbances, increased by closure of the eyes, and, furthermore, since every other possible factor had been eliminated we must conclude that the equilibrium disturbance was due to a loss of some organ of equilibrium located in the inner ear.

Finally, if the loss of equilibrium was due to destruction of something within the inner ear and the tests employed were not rotational (thus excluding the semicircular canals), then it follows that the lost function must have been due to the destruction of the vestibular sacs. The writer in his work on labyrinthine equilibrium disturbances declared in favor of the Breuer theory, which in part declares the functions of the utriculus and sacculus to be that of perception of straight line (progressive) movements, allowing the functions of the semicircular canals to be for the perception of turning movements.

THE WRITER'S CONCLUSIONS.

The functions of the utriculus and sacculus are both static and dynamic.

Static—for the perception of position in space.

Dynamic—for the perception of straight line (progressive) movements in any direction, so long as the movement is increasing or diminishing in velocity.

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33. Von Stein—"Ueber Gleichgewichtsstoerungen bie Ohrenleiden"—Zeitschn. f. Ohren., Bd. 27, 1895.

34. Pollak—"Ueber galvanischen Schwindel bei Taubstummen und seine Beziehungen zur Function des Ohrenlabyrinthes"—Pfluegers Arch., Bd. 54, 1893.

35. Kreidl—"Beitraege zur Physiologie des Ohrlabyrinthes auf Grund von Versuchen an Taubstummen"—Arch. f. Ohrenheilk, Bd. 51, 1891.

36. Alexander and Mackenzie—"Functionspruefungen des Gehororganes an Taubstummen"—Monatschrift fuer Ohren., 1908, und Zeitschrift fuer Ohren., 1908.

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VERTIGO.

THE subject of vertigo has become one of importance, especially in view of the recent advances made in our knowledge of the physiology and pathology of the ear labyrinth.

Although vertigo in conjunction with nystagmus and equilibrium disturbances has been referred to in every recent publication upon the labyrinth and diseases of the labyrinth, writers as a rule have not attempted to make clear to the minds of their readers exactly what is meant by the term. As a result, not only the laity but some physicians have but a vague conception of the symptom. I find this especially so when noting the history of the cases. Of all those who claim to have vertigo, upon close questioning I find that many have not. I am, therefore, prompted to write a paper upon this subject.

WHAT IS VERTIGO?

I agree, in substance, with Panse that vertigo in its broadest sense is that sensation of confusion which results from any false perception of one's relative position to space or motion in space.

Normally one is aware of the fact whether he is standing vertically erect, inclined forward, backward or to either side, sitting or lying down; whether he is moving in a straight line forward, backward or to either side, vertically upward or downward; whether he is moving in a circle in any of the three principal planes or resultant of these planes.

Furthermore, one is aware of the fact whether surrounding objects are out of plumb, so to speak, inclined toward or away from him, to one or the other side. Finally one is able to determine whether objects are stationary or in motion, and when in motion whether they are moving toward or away from him, to one or the other side, vertically upward or downward, in a circle in front of him etc.

We are made conscious of these positions and motions, subjective or objective, through three different peripheral sense organs and their centripetal tracts. 1.—Thru the visual organ, the eyes, including their intrinsic and extrinsic muscles; 2.—Thru the equilibrium sense organs, the nonacoustic labyrinth; 3.—Thru the kinesthetic sense organs; the deep muscles, joints, sensory nerve endings all over the

body, which may, since their functions are identical, be classed as one organ.

The normal, physiologic, correllation of these three centripetal impulses from the eyes, ears (nonacoustic labyrinth) and muscles and joints, and possibly the skin and viscera, enables one to maintain perfect equilibrium of the body when at rest (static) and during motion (dynamic).

According to Bechterew, quoted by Panse, the correllation of all these centripetal impulses takes place in the cerebellum. Any *sudden* falsification of sensation from one of these organs—whether produced experimentally or pathologically—produces that unpleasant sensation of confusion known as vertigo and in proportion, equilibrium disturbances. However, a falsification of sensation from any one of these three sensory organs if continued will eventually be corrected by the remaining two. With this correction comes a relief of the vertigo. In other words, the contradictory or false sensation will be suppressed while the remaining virtual sensations will be accepted.

These above conditions are true only in so far as they apply to cases of permanent and fixed falsification; for instance: in cases of labyrinth suppuration, those of permanent eye muscle paralysis, etc. On the other hand, vertigo *may* recur in cases of variable falsification, for instance in cases of irritative lesions or in those cases of partial loss, followed by subsequent restitution of function. In cases of temporary eye muscle paresis when the affected muscle begins to functionate again we may find vertigo accompanied with nystagmus during attempts to roll the eye in the direction of the muscle's action; observed by Stewart (*Deutsches Medizin. Zeitschr.*, 1895, seite 511) and others, including the writer.

While vertigo is always accompanied by pronounced equilibrium disturbances the reverse is not true.

At this writing I wish to deny the claim of some authors, quoted by the writer in one of his recent papers upon labyrinth suppuration, that vertigo is always associated with nystagmus.

Although vertigo may disappear after a few days in cases of permanent loss of function of one of these end organs and the equilibrium disturbances may become less pronounced, there still remains a slight degree of *permanent* equilibrium disturbance. This is theoretically, mathematically and practically so as shown by the writer in a recent

paper upon the subject of equilibrium disturbances, entitled "Klinische Untersuchungen ueber die labyrintharen Gleichgewichtsstoerungen mit besonderer Berucksichtigung der allgemeinen Pruefungsmethoden und des Gonimeters" (*Archiv. f. Ohrenhlk.*, band 78, Feb., 1909.)

If three different sets of centripetal impulses are necessary for perfect orientation and equilibration, it must follow that when one of these is lost, orientation and equilibration must be disturbed in proportion; however, with the remaining two impulses one learns rapidly to orientate and equilibrate, quite sufficiently for ordinary purposes. In fact one may get along so well under favorable circumstances as to almost conceal the presence of his equilibrium disturbance; but careful tests of such patients by Von Stein, Krotoscheiner, Kreidl, Alexander and Mackenzie have never failed to prove their presence.

When a second of these centripetal impulses is lost, naturally orientation and equilibration are disturbed still further, and proportionately more than when the first impulse alone was lost.

Sudden falsification of sensation from one of these three peripheral sense organs leads not only to vertigo, as already stated, but also to pronounced equilibrium disturbance, nausea and vomiting, vasomotor changes—flushing or more frequently pallor of the face—profuse sweating, etc.

The sensation and intensity of the vertigo differs in different cases according to which of the three special end organs or centripetal tracts is involved.

In the case of vertigo resulting from experimental irritation of, or pathologic lesion in, the labyrinth or its centripetal tracts, the vertigo is always of the type called rotational; that is to say, the patient suffers either the sensation as though external objects were in motion or else the sensation of subjective motion. Whether the sensation of motion is referred to external objects or to the patient himself, these motions are in circles in one of the three principal planes or resultant of these planes.

Rotational vertigo is perhaps the most intense and unpleasant form and is always combined with nystagmus.

While vertigo the result of affection of the labyrinth or its centripetal tracts is always rotational in character and is combined with nystagmus, it does not follow that every rotational vertigo combined with nystagmus must necessarily be produced by disease of the laby-

rinth or its centripetal tracts; for on the contrary we find rotational vertigo and nystagmus in cases of vertigo produced by a lesion in the eye—or, more exactly, in cases of eye muscle paresis during the stage of improvement (Panse). However, as a rule rotational vertigo indicates a lesion in the labyrinth its centripetal tracts or their terminals in the cerebellum.

In the case of vertigo resulting from disease of the eye or eye muscles, excepting in the case above mentioned, the patient's sensation of vertigo is referred to external objects; they seem too near or too distant, inclined toward or away from him to one or the other side, or the floor upon which the patient is standing is inclined in one of these directions, etc., and on account of which the patient is in danger of making false steps and movements. He misjudges the distances and directions of objects.

This ocular form of vertigo is less intense as a rule than the vestibular form and is unassociated with nystagmus except in the case previously referred to.

Ocular vertigo is characteristically ameliorated by closure of the eyes, whereas the former vestibular or rotational is uninfluenced. This is quite an important differential test for these two forms.

In contrast with the ocular vertigo we have the vertigo due to falsification of centripetal impulses from the muscles, joints, etc., (the so-called deep sensibility). In this form of vertigo, for we may call it so—accepting the broad definition, the patient perceives the external world in its normal relations, but suffers the subjective sensation as though he himself was out of plumb with it. He feels as though he is tilted forward, backward, or to one or the other side, or else when making a movement he believes he has made it either too great or too slight; in other words, he is not properly informed of either the extent or character of the movement, his perception of the movement (excepting when aided by the eyes) causes him to innervate too strongly or not strongly enough; he makes incorrect movements believing he has made them correctly.

In contrasting the kinesthetic form of vertigo with the ocular it may be said that while ocular vertigo is improved by closure of the eyes, the kinesthetic form is made worse and its accompanying phenomenon equilibrium disturbance, is decidedly aggravated.

Let us consider next some of the more definite causes of and the sensations produced by these three separate forms of vertigo.

I. **Rotational or vestibular** vertigo may be produced experimentally or pathologically. *Experimentally* by:

(a) turning (as first practiced by Flourens) in a revolving chair. This should be done with the patient's eyes closed during the turning in order to obtain a pure vestibular form, otherwise a combination of vestibular and the ocular is produced.

(b) caloric irritation of the nonacoustic labyrinth.

(c) galvanism with opposite electrodes applied to opposite sides of the head or with one electrode applied to the region of the ear and the other applied to some other part of the body, say the hand.

(d) mechanical irritation of the semicircular canals after the manner of Ewald's experiment.

(e) sectioning one or both of the 8th nerve stems.

(f) removal of a cerebellar hemisphere. These latter three experiments are not applicable to human subjects.

Pathologically by:

(a) any lesion in the nonacoustic labyrinth: including congestion, circumscribed irritative or destructive lesions, diffuse serous or suppurative inflammations, toxemias, growths, etc.

(b) any lesion in the vestibular nerve: neuritis, tumors, basal fractures, meningitis, etc.

(c) any intracranial lesions involving the nuclei or tracts to the cerebellum or the cerebellum itself.

In the case of vertigo produced by any of these causes, experimental or pathologic, the sensation of subjective or objective turning is produced. The vertigo persists even with the eyes closed.

With the eyes closed the sensation is that of subjective turning; the patient feels as though he is being turned. With the eyes open the sensation is generally that of objective turning, the patient feels as though surrounding objects were turning. The direction and plane of the turning is dependent upon the direction and plane of the nystagmus which is constantly present in this form of vertigo.

The equilibrium disturbance manifests itself by falling or turning in the opposite direction to and in the plane of the nystagmus. This falling results from the efforts of the patient to correct the sensation of apparent falling. Like the sensation of vertigo, the equilibrium disturbance is manifest even more with the eyes closed than when open. Again by the aid of touch and the deep muscle sense, the pa-

tient is aided in maintaining equilibrium; which accounts for the fact that patients suffering with vestibular vertigo immediately grasp, for support, the nearest fixed object.

II. **Ocular vertigo** may be produced experimentally or pathologically. *Experimentally* by:

(a) sudden disturbance of vision, especially when monocular; for instance by the placing of strong concave or convex spherical lenses before one or both eyes or the placing of a minus glass before one eye and a plus glass before the other. The convex glass, because of the magnification, produces the sensation of nearness of objects while the indistinctness of the outlines and details produces the contradictory sensation of remoteness. The concave glass produces just the opposite contradiction of sensations.

(b) the placing of plus or minus cylinders before the eyes produces distortion of objects, irregularity in angles, etc.; for instance, right angles are made to appear too acute or too obtuse, dependent upon whether a plus or minus cylinder is used and the axis to which it may be turned.

(c) sudden paralysis of accommodation with dilatation of the pupil produced by a mydriatic will cause vertigo, especially in those suffering from considerable latent hyperopia or hyperopic astigmia. The patients complain usually of objects, especially the floor, being too near or distant, dependent upon the amount and axis of the manifest astigmia.

(d) The placing before the eyes of prisms, whether strong enough to produce diplopia or not, will cause vertigo. The placing of prisms, bases in the same direction, produces slanting effects of external objects and false impression of location. The placing of the bases in opposite directions, if strong enough, produces diplopia with vertigo. On the other hand, the use of weaker prisms, insufficient to produce diplopia, produces a vertigo often more intense than that produced by the stronger prisms. With the weaker prism producing vertigo, there is always associated nystagmus directed toward the apex of the prism. In the case of vertigo produced by prisms, the patient is confused most as to the direction of external objects.

(e) Tenotomy or section of the nerve supply to one of the extrinsic muscles produces similar effects to that produced by prisms.

(f) Pressure of the finger upon the one eyeball may so change the direction of the visual axis as to produce diplopia with vertigo, etc.

(g) There is a form of vertigo combined with nystagmus, which we may conveniently term visual rotational vertigo and may be produced experimentally by gazing for a long time at moving objects—waterfalls, rapidly moving currents, carousal, windmills in motion—or by Mack's rotating cylinder, etc. After looking for a long time at one of these moving objects and then looking away at stationary surroundings the patient suffers the sensation of a mild rotational vertigo, whereby the stationary objects appear to be moving in a contrary direction, and when this vertigo is pronounced it is accompanied by equilibrium disturbance characteristic of rotational vertigo. This vertigo is similar to the vertigo associated with the socalled after-turning nystagmus; the character of the vertigo might suggest the presence of rhythmic nystagmus, but up to the present time the writer has been unable to demonstrate it.

On the other hand an experiment practiced by the writer would suggest the absence of nystagmus. The experiment is made as follows: Seated on the rear end of a moving train (observation platform) one directs his view straight ahead at some distant receding point for about 30 to 40 seconds. This produces no nystagmus, but naturally the sensation as though the field of vision was receding. Should the train come to a stop, or should one direct his gaze at some stationary object (within the car) the sensation of reversed motion is produced lasting for several seconds. This is an ocular form of after-motion vertigo, and is unassociated with nystagmus. This visual sensation of reversed motion is furthermore unassociated with any labyrinthine function from the fact that it can be produced only with the eyes open.

Pathologically by:

(a) Sudden irregular swelling of the lens, as sometimes found in incipient cataract, may cause polyopia with unpleasant vertigo.

(b) Sudden blindness from a lesion in the perceiving apparatus, retina and optic nerve—tho it causes a loss of the perspective sense seldom if ever produces unpleasant vertigo, from the fact that we have long since learned to accommodate ourselves to monocular vision. The nail driving experiment with one eye closed is familiar to you all.

(c) Lesions especially in the short optic tracts to the ganglion genicula externa, corpora quadrigemina anterior and the pulvinar—and perhaps too, in a measure, the long tracts to the visual centers in

the occipitial lobe, produce not infrequently vertigo with equilibrium disturbances—according to Bechterew (*Deutsche Med. Zeitung,* 1894, No. 46). These are produced in the former instance by disturbances more especially in the pupillary and accommodation reflex arc and in the latter instance by disturbance in visual perception.

(d) Sudden paralysis of accommodation from a lesion in the ciliary muscle itself, nerve supply, ciliary ganglion, that part of the oculomotor nucleus which controls accommodation, produces the sensation of vertigo with equilibrium disturbance, similar to that produced by mydriatics.

(e) Sudden paralysis of one or more of the extrinsic eye muscles from lesion in the muscle, nerve or nucleus, produces diplopia with vertigo.

The sensation as well as the mechanism of vertigo produced by *complete paralysis* of one of the extrinsic eye muscles differs from that produced by *slight paresis.*

In case of paralysis, the patient manifests real squint but no nystagmus; the patient suffers diplopia and a sensation of confusion (vertigo) as to the location of external objects, especially objects located to the side or direction of the muscle's action. For example: in case of paralysis of the right external rectus, the location of objects to the right are estimated to be farther to the right than they actually are. The reason is that since we have learned to estimate the location of objects by the amount of muscle innervation necessary to fix the objects for central vision, any extra amount of innervation put forth in the attempt to fix them is interpreted by the patient as an extra amount of angle deviation of the object to the right. This is associated with a corresponding amount of secondary deviation of the fellow eye to the right. Such a patient in attempting to grasp an object lying to the right of him will invariably reach too far to the right. In brief, there is confusion as to the location of external objects.

In the case of slight paresis, or in those cases of late paralysis where a restoration of function has begun in the affected muscle, the sensation and mechanism of the vertigo is quite different. In these cases the patient may manifest but little or no squint, but instead will show rhythmic nystagmus, especially when looking in the direction of the muscle's action. The vertigo is present only when looking in the direction of the paretic muscle, is rotational in character and associated

with equilibrium disturbance corresponding to rotational vertigo. This trio of symptoms—rhythmic nystagmus, vertigo and equilibrium disturbance—may simulate closely the vestibular form; the nystagmus of eye muscle paresis is more likely to be unilateral, and less intense than in the case of nystagmus of the vestibular form. Again, the vertigo of eye muscle paresis differs from that of the vestibular form in that the vertigo improves upon closure of the eyes; whereas the vestibular form does not.

III. **Kinesthetic vertigo.** That vertigo may be produced by falsification of sensory impulses from the muscles, joints, etc., may seem to some far fetched; nevertheless the fact is true if we accept the broad definition of the term offered in the early part of the paper. For instance:

The vertigo resulting from paresis of the eye muscles is produced by two factors: (1) visual (diplopia) and (2) muscular (falsification of sensation of the affected muscle). The concomitant equilibrium disturbances would indicate that the vertigo results more from the falsification of sensory impulses from the muscles than from the diplopia. The author has arrived at this conclusion from the results obtained by equilibrium tests made upon patients suffering from paralytic squint. These patients were examined (1) with both eyes open; (2) with the affected eye closed, and (3) with the well eye closed, the patients being directed to look in the direction of the action of the paralyzed muscle.

With both eyes open, the gait and equilibrium were disturbed.

With affected eye closed, the gait and equilibrium were quite normal.

With the well eye closed, gait and equilibrium were disturbed and quite as markedly so as when both eyes were open.

Kinesthetic vertigo may be produced experimentally or pathologically. The production of *experimental* kinesthetic vertigo has been only partially successful, from the fact that the deep muscle and joint sensibilities are so widely distributed over the entire body as to make it quite impossible to irritate or anesthetize a sufficient proportion of them to produce marked vertigo and equilibrium disturbance. The results of experiments thus far made have been sufficiently successful however to warrant further experimentation after more improved methods. A few of these experiments may be mentioned:

(a) Anesthesia of the soles of the feet produced artificially has re-

sulted in the subject manifesting positive Rhomberg sign and disturbed gait (Vierordt's experiment quoted by Bechterew. *Pfluger's Arch.* XXX, s. 517).

(b) Experiments by Mach with the two feet placed air tight in two holes in a box resulted in the sensation as though the floor was elevated when aspiration of air from the box was made.

(c) Further experiments by Mach with weights (vessels containing water) held in the hand or attached to the shoulders caused the sensation as though the arms or body were elevated when the load was removed.

(d) Panse tells of an interesting experiment made by Mach, as follows: two vessels so constructed as to permit a gradual emptying when filled with water, were adjusted to a cap arrangement fitted on the head; this when set in horizontal rotation, produced, as the weight diminished from loss of water, the subjective sensation of turning in the opposite direction.

(e) Bechterew (*Pfluger's Arch.* XXXIV) found that section of the posterior cerebellar peduncle caused animals to roll to the side operated.

Kinesthetic vertigo with equilibrium disturbance may be produced *pathologically* by:

(a) Acute polyneuritis, involving sensory nerves.

(b) Diseases involving the posterior columns, Gall and Burdach or spinocerebellar tracts in the cord, tabes, Friedrich's disease, multiple sclerosis of the cord, hemorrhages and myelitis in the cord, etc.

(c) Diseases in the corpus restiforme, interrupting the centripetal impulses.

In all these and others not mentioned the equilibrium disturbance is a more pronounced symptom than the vertigo, and lasts permanently in cases of permanent destructive lesions. Furthermore, the equilibrium disturbance from interruption of these impulses is even more pronounced than in the case of interruption of impulses from the eye while the vertigo is less pronounced.

Vertigo is never complained of as being so unpleasant as in the case of interruption of impulses from the eye or ear. This is due to two circumstances: (1) That the disease which produce interruption of sensation from the muscle and joint sense endings is very rarely acute, except in the case of acute hemorrhagic lesions; and (2) That these diseases are very rarely extensive enough at the onset. On the con-

trary, they are usually gradual progressive conditions which permit the patient to become gradually accommodated to the changes.

The vertigo and equilibrium disturbances, like those of vestibular origin, are exaggerated by closing the eyes.

CENTRAL VERTIGO.

Finally we come to a discussion of that large class of so-called central vertigoes.

By central vertigo is not meant the vertigo which results from anatomic changes in the brain substance itself, which presents, according to the location of the lesion, the characteristic of *one* of the three definite types already referred to; but on the contrary, by central vertigo is meant that form of vertigo which is due to circulatory disturbances in the brain or to toxic substances circulating in the blood current; and it presents a combination of the *several* characteristics of all three types. In other words, central vertigo differs from each of the three described types—or primary forms—in that the manifestations in the primary forms are distinct and definite; while in the central forms they are not.

That central vertigo should present the combined features of all three primary forms, but less defined and distinct than any one of them is quite evident when we recall the etiology.

In the primary forms we have a vertigo due to a well defined exaggeration or suppression of the normal impulses from one of the three peripheral orientation organs through an irritative or destructive lesion in these organs or their centripetal tracts, while in the central vertigo due to circulatory disturbances the exaggeration or suppression of impulses are more general and less pronounced; for instance, in the case of congestion or anemia of the brain the whole brain suffers whereby all centers are more or less affected. Again, in the case of toxemia the toxic substance circulating in the blood current is never so selective in its action as to affect the centers for one of these impulses to the exclusion of the remaining two. However, exceptional cases do occur where the toxic substance may be sufficiently selective in its action as to affect certain centers more than others when the resulting vertigo may present the characteristics of one primary form more prominently than the other two. In such cases a differential diagnosis of the central vertigo from the primary forms, which it resembles, may be quite

difficult. A similar condition may result in cases of arteriosclerosis or endarteritis where certain vessels may be more affected than others—or where the blood vessels make a sharp turn, or in cases where vessels branch off from the main stem at a sharp angle.

Central vertigo may be produced by the following familiar conditions:

I. Cerebral congestion, active or passive, from any cause

II. Cerebral anemia.—Psychic or physical shock producing syncope. Insufficiency of the heart's action. Post-hemorrhagic anemia. General anemia, including chlorosis, pernicious anemia, etc. Changes in the blood vessels narrowing their lumen; arteriosclerosis, endarteritis, etc.

III. Toxemias, acute or chronic.

Acute: Tobacco, alcohol, carbon dioxide, ether, chloroform, benzine and the inhalation of other volatile toxic substances.

Chronic: Tobacco, alcohol, lead, etc. Uremia, autointoxication. Toxemias associated with the acute infectious fevers, etc.

All vertigoes are aggravated by change of body position, especially from lying down to standing up. This aggravation is more pronounced in the case of vertigo from cerebral anemia than in the case of vertigo from any other cause. This has been felt at some time in the experience of most of us, especially after a depleting illness, which at the same time required us to remain in bed for some days or weeks.

Concerning vertigo of gastric origin the writer is quite skeptical. It is true that the ingestion of certain toxic substances produces vomiting with vertigo, but in these cases the author believes that the vomiting is merely a symptom of the vertigo, and the toxic substances ingested would produce vertigo at all events even though the patient did not reach a stage where vomiting resulted. If irritation of the pneumogastric was the cause of the vertigo then it must follow that all substances which are capable of producing emesis should at the same time produce vertigo. Since this is not the case, but the reverse is true, then it follows that nausea and vomiting are the results and not the cause of the vertigo.

LABYRINTH FISTULA.

WHAT IS A LABYRINTH FISTULA?

A LABYRINTH fistula consists of any loss of substance in the osseous labyrinth which permits an artificial path of communication between its cavity and the middle ear space. They vary in size, shape and location; generally occur as single perforations, but may be found multiple.

The most frequent locations of labyrinth fistula are, naturally the most exposed, and at the same time most vulnerable, points in the lateral labyrinth wall; namely—the prominence of the external semicircular canal, the promontory, the oval and round windows; however, no part of the osseous labyrinth is entirely exempt. Fistulæ of the labyrinth have been found in the region of the external crus of the superior semicircular canal; besides, cases have been reported of the inner labyrinthine wall (by Koerner, Jansen and Habermann) resulting from long standing deep extradural abscess; while in cases of necrosis. the loss of substance in the labyrinth capsule may extend in almost any imaginable direction.

ETIOLOGY AND PATHOLOGY.

Since labyrinth fistula is a process secondary to middle ear suppuration and more especially that of the chronic form, we must, in seeking for the etiology of labyrinth fistula, seek for the etiology of the primary middle ear suppuration and especially for those causes which tend to produce chronicity of an already existing acute suppuration.

I shall not take the time here to discuss all the predisposing and active causes of chronic middle ear suppuration, but proceed immediately to a brief discussion of the more direct causes which tend to produce fistulæ; among these may be mentioned:

(1) Cholesteatoma. This is pre-eminently the most frequent of the direct causes of labyrinth fistula. By its growth that part of the lateral bony labyrinth wall with which it comes in direct contact is gradually absorbed until a saucer shaped fistula results.

(2) Adhesive bands between the meso- and epitympanic spaces or antrum leading to the formation of closed or nearly closed cavities

which favor retention of secretion and pressure. The pressure of the confined secretion leads to a destruction of the bone through caries or necrosis or both combined.

(3) Polyps and granulations. These may, by reason of their presence, operate in a similar manner as do the adhesions—to produce retention with pressure resulting in the destruction of the bone. Granulations in themselves are generally an evidence of deep seated bone involvement (osteitis) more often of the rarifying type. Granulations occur frequently in the depressions about the windows and lead eventually to destruction of the periosteum, thrombi of the nutrient vessels of the underlying bony capsule with resulting caries and necrosis.

(4) Local tuberculosis. This comprises a destructive form of chronic middle ear suppuration liable to result in fistula of the labyrinth by reason of infiltration of the periosteum with subsequent softening, detachment of the periosteum and devitalization of the bone.

(5) Diabetes. This condition especially favors necrosis, sequestra partial or complete of the labyrinth capsule being quite common.

(6) Prolonged constitutional diseases which lower the vitality of the patient or impair the power of repair.

(7) Finally, unhygienic surroundings, poor nourishment and neglect of proper treatment may be mentioned; however, these last three belong more to the indirect or contributory causes.

PATHOLOGY.

The microscopic pathology of labyrinth fistula will not be discussed in this paper for the reason that (1) the microscopic pathology differs considerably in different cases, depending principally upon the cause; (2) a sufficient number of cases have not yet been studied to warrant a thorough discussion of it; (3) the intention of the writer is to discuss rather the clinical pathology. According to the extent and depth of the destructive process which produced the fistula, we may find any one of the following conditions of the membranous labyrinth present:

1, Normal and intact. 2, Circumscribed irritative process (congestion). 3, Circumscribed destructive process (circumscribed suppuration or granulations). 4, Diffuse destructive process (diffuse labyrinth suppuration). Chronic diffuse plastic or obstructive inflammation of the membranous labyrinth will not be considered because (1) of its rare association with fistula; (2) from the fact that it does not afford us a

distinct clinicopathologic type; (3) its discussion here would lead to too much confusion in the grasping of the more frequent and fundamental conditions.

If one is able to comprehend these four fundamental conditions, he will be in a position to work out a clinical diagnosis of the rarer complex conditions. These four conditions may be considered the four stages of the same process which follow each other in the order above mentioned. The transition from one stage to the next succeeding may take place rapidly or slowly, or there may be a prolonged or even permanent arrest of the process at any stage. This arrest is especially apt to take place during the third stage so that the fourth stage may never be reached. In some cases, especially where the circumscribed destructive lesion is located in the horizontal semicircular canal, the spontaneous arrest of the process in the third stage is very desirable, since the patient retains fair hearing. For this reason our efforts should be to treat such cases with this object in view.

These four conditions of the membranous labyrinth, three of which are pathologic, associated with fistula give rise to four separate clinical pictures. The first three of these conditions we are able to diagnose and differentiate, but the fourth condition is really one of the forms of labyrinth suppuration which is impossible of differentiation from the other forms of labyrinth suppuration without fistula; and from the standpoint of prognosis and treatment the presence or absence of a fistula plays no important role.

In order to better understand the symptoms and signs of labyrinth fistula when we come to discuss them, it is well that we digress for the present and consider briefly Ewald's physiologic experiments upon the semicircular canals and Gellé's test of the acoustic function.

Concerning Ewald's experiment you are referred to the paper on "Nystagmus."

The clinical application of Ewald's experiment, but slightly modified, may be applied as an examination test for the diagnosis of fistula of the static labyrinth.

Since in most cases we are unable by otoscopic examination to actually see the fistula and apply compression and aspiration directly to the membranous labyrinth, we are compelled to do so indirectly by increasing and diminishing the atmospheric pressure in the tympanic cavity. This is accomplished by a simple device consisting of an olive

shaped hard rubber tip fitted on the distal end of a soft rubber tube, the proximal end of which is attached to a Politzer balloon, a Gellé balloon or any other suitable force and suction apparatus.

The olive tip is introduced into the external auditory canal air tight and may be held in place by the patient; the physician is thus free to manipulate the balloon with one hand and to elevate the upper eyelid with the thumb of the other hand. A positive fistula sign is manifested by a pronounced nystagmus in one direction when the compression of the balloon is made, and a less pronounced nystagmus in the opposite direction when aspiration is made. Again, both compression and aspiration nystagmus are accompanied by more or less marked vertigo.

A negative fistula sign is manifested by the absence of these phenomena.

The plane and direction of the compression and aspiration nystagmus varies with the location of the fistula. A horizontal nystagmus to the same side by compression and the opposite side by aspiration is the m o s t f r e q u e n t and at the same time the m o s t p o s i t i v e indication of fistula and that of the horizontal semicircular canal.

A rotatory nystagmus to the opposite side by compression and to the same side by aspiration indicates generally but not always a fistula of the superior semicircular canal. At one time Neumann, and likewise the writer, believed that this nystagmus indicated a fistula of the external vestibular wall or loss of the stapes plate, but subsequent events have not verified this supposition.

A rotatory nystagmus to the same side by compression and to the opposite side by aspiration may indicate a fistula of the external vestibular wall or of the stapes plate, but not absolutely.

In brief the most certain localizing compression and aspiration nystagmus is in the case of the external semicircular canal. In all other cases of compression and aspiration nystagmus we can say, at most, that the signs indicate fistula of the static labyrinth capsule but the exact location in all cases, for the present at least, remains more or less an open question.

The question arises: Is the so-called compression and aspiration nystagmus an infallible sign of fistula? The answer must be "no," from the fact that we find cases where the sign is negative in spite of the presence of fistula, and on the other hand we find cases where the

sign is positive without the actual presence of a fistula. The reasons for these discrepancies have been pointed out by Alexander and Lasalle, "Ueber den durch Luftdruckveränderungen auslösbaren Nystagmus und das Fistelsymptom, Wiener klin. Rundschen, 1908, and by the writer. The reasons are as follows: —

I—Negative fistula sign with positive fistula may be found:

(a) In cases of labyrinth destruction (suppuration); since the labyrinth is nonreactive the sign must be negative.

(b) In cases of obstruction from polyps, adhesions or large cholesteatoma plugging up the fistula, compression of the membranous labyrinth is quite impossible.

(c) In cases of large dilated Eustachian tubes, the air escapes thru the open tube and makes impossible any increase of atmospheric pressure within the middle ear spaces.

II—Positive fistula sign with negative fistula may be found:

(a) In cases of acute middle ear inflammation (frequently observed by the writer). The explanation offered is that of a combination of factors; first, the Eustachian tube is closed by inflammatory swelling; second, because of this closure the secretion is more confined to the middle ear and cannot escape thru the tube; third, compression of the secretion acts as a hydraulic pressure which is necessarily greater than pneumatic pressure; fourth, the ligament surrounding the stapes is more mobile because of the presence of secretion than it normally is when there is no secretion in the middle ear space.

(b) In case of chronic middle ear suppuration with loss of the hammer and incus (observation by Alexander).

Notwithstanding the fallibility of the fistula sign, the presence of it, especially when marked, is strongly suggestive of labyrinth fistula.

GELLE'S TEST.

Gellé's test was originally intended as a test for the determination of ankylosis of the stapes in the oval window in cases of otosclerosis. The test is made with an apparatus similar to that used for the determination of compression and aspiration nystagmus (described above) with the exception that compression only is used.

Normally compression of air in the external canal is communicated to the stapes, which is forced inward and held so by the pressure from the tympanic side. The effect of the forcible pressure inward of the

stapes plate is to produce impairment of hearing of a type corresponding to disease of the perceiving apparatus; namely, Rinné positive with shortened bone conduction. Weber lateralized to the opposite side, etc. This effect upon the normal ear is known as a positive Gellé sign.

In cases of otosclerosis the compression of air in the canal produces no effect upon the hearing, since the stapes plate is ankylosed in the oval window. This negative effect is designated "negative Gellé sign."

In cases of fistula or abnormally loose and movable stapes, instead of the normally moderate positive Gellé we find abnormally pronounced positive Gellé sign, which we may term a fistula sign of the acoustic labyrinth. According to Politzer, in cases of pathologic condition of the membranous labyrinth the positive Gellé sign is likely to be more pronounced than in those cases with normal membranous labyrinth.

The principle of the Gellé test may be applied in one of two ways: *First*—after inserting the olive tip air tight in the canal, apply the handle of a vibrating tuning fork (Politzer middle tone) to the bag. When compressing the bag with the hand the pressure of air in the ear is increased and upon relaxation the pressure is diminished. With the normal ear the fork is heard less distinctly during compression than during relaxation; this is recognized as a normally positive Gellé. In the case of otosclerosis, where the foot plate of the stirrup is ankylosed in the oval window, the compression of air does not produce the same degree of diminution of hearing as the normal; this is recognized as a negative Gellé.

In the case of abnormally loose stapes or where a fistula exists in the region of the vestibule, the diminution of hearing is greater than normal; this is recognized as an abnormally positive Gellé.

Secondly—Since compression of air in the ear presses the foot-plate of the stapes inward in the oval window an experimental inner ear disease is produced in normal individuals, therefore it follows that the vibrating fork held to the mastoid is heard less distinctly or not at all during the compression of air in the ear and heard again during relaxation.

In the case of ankylosis of the foot-plate in the oval window the diminution of bone conduction is less evident than normal, and in the case of abnormally loose stapes plate or in case of fistula this diminution of bone conduction is more evident than normal.

By a comparison with a sufficient number of normal cases one learns to recognize the difference between the normally positive Gellé and the abnormally positive as found in fistula of the acoustic labyrinth.

SYMPTOMS AND SIGNS OF LABYRINTH FISTULA.

I—**With normal and intact membranous labyrinth.**

1. By otoscopic examination evidence of middle ear suppuration, *i. e.*, partial or complete destruction of the tympanic membrane, discharge, etc.

2. Impairment of hearing of the type belonging to disease of the conducting apparatus: Weber to diseased side, lengthened bone conduction, negative Rinné, etc.:

3. Absence of acoustic and static labyrinth symptoms including tinnitus, vertigo, nystagmus and equilibrium disturbances

4. Normal reactions of static labyrinth to turning, caloric and galvanic irritations.

5. Positive fistula sign.

(a) If the fistula is located in the region of the static labyrinth, we find marked compression and aspiration nystagmus associated with vertigo (making due allowance for the exceptions already mentioned).

(b) If the fistula is located in the region of the acoustic labyrinth we find a very positive Gellé sign. In some rare cases with a single fistula located in the region of the vestibule we find a combination of these two fistula signs.

II—**Labyrinth fistula with circumscribed irritative process of the membranous labyrinth.**

According to the location of the fistula there may be present one of two distinct and separate pictures; they are:

(A) In case of fistula of the acoustic labyrinth with irritative process of corresponding part of the membranous labyrinth:

1. By otoscopic examination evidence of middle ear suppuration as in other cases of labyrinth fistula.

2. Impairment of hearing of the type belonging to disease of the conducting apparatus with a slight element belonging to disease of the perceiving apparatus.

3. Tinnitus corresponding in pitch to the location in the cochlea which is involved.

4. Absence of static labyrinth symptoms; vertigo, nystagmus, equilibrium disturbance.

5. Reaction of the static labyrinth normal.

6. Very positive Gellé symptom. In other words, by compression abnormally marked diminution of hearing, especially thru the bone.

(B) In case of fistula of the static labyrinth with irritative process ot the corresponding part of the membranous labyrinth we find:

1. By otoscopic examination, evidence of middle ear suppuration as in other cases.

2. Impairment of hearing of the type belonging to disease of the conducting apparatus.

3. Absence of acoustic labyrinth symptoms, or more concisely speaking, there is no tinnitus.

4. Positive static labyrinth symptoms:

(a) Vertigo pronounced or slight, depending upon the degree and extent of the process and characteristically aggravated by rapid movements of the head, no matter whether they are made actively by the patient or passively by the surgeon.

(b) Nystagmus, rotatory, horizontal or mixed, to the d i s e a s e d side. The nystagmus may be constant or intermittent and like the vertigo can be excited by the surgeon making rapid passive movements of the patient's head.

(c) Equilibrium disturbances which have been amply described in the series of papers upon labyrinth suppuration and elsewhere and can be easily determined by Stein's, Alexander's and Mackenzie's tests.*

5. Reactions of static labyrinth i n c r e a s e d upon the diseased side to turning and to the caloric and galvanic irritations.

6. Positive fistula sign; that is, marked compression and aspiration nystagmus.

III—**Labyrinth fistula with circumscribed destructive process in the membranous labyrinth,** like the former condition, may present one of two distinct clinical pictures according to the location of the fistula. They are:

(A) In case of fistula of the acoustic labyrinth with destructive process of the corresponding part of the membranous labyrinth, the symptoms and signs are:

1. By otoscopic examination, evidence of middle ear suppuration as before.

2. General impairment of hearing of the type belonging to disease of the conduction apparatus, together with absolute deafness for certain

tones corresponding to the area of destruction. In other words—single or multiple scotoma. This latter circumscribed deafness is of the type belonging to affections of the perceiving apparatus. These may be best ascertained by the use of Bezold's continuous chain of forks.

3. Tinnitus is present during the active stage since there is a zone of irritation surrounding the area of destruction. In a very late stage tinnitus may be absent.

4. Absence of static labyrinth symptoms.

5. Static labyrinth reacts normally to the usual examination methods.

6. Gellé's sign may be positive or negative, depending upon the size of the fistula and the extent and character of the destruction.

In those cases where it is possible to produce compression of the endo- or perilymph beyond the areas of the destructive process, the Gellé sign would be proportionately positive; on the other hand, in those cases where extensive granulations or organized exudate within the labyrinth prevent the possibility of compression of the endo- and perilymph beyond the area of the destructive process, the Gellé sign must be negative.

(B) In cases of fistula of the static labyrinth with destructive process of the corresponding part of the membranous labyrinth, the symptoms and signs are:

1. By otoscopic examination, evidence of middle ear suppuration as before.

2. Impairment of hearing of the type belonging to disease of the conducting apparatus.

3. Absence of tinnitus.

4. Positive static labyrinth symptoms: (a) vertigo is always present in acute cases, absent in late cases. The vertigo is aggravated by rapid head movements; (b) nystagmus, rotatory, horizontal or mixed, to the unaffected side. The nystagmus is more constant, but rapidly diminishes in intensity and may eventually disappear; (c) equilibrium disturbances are pronounced in early stages and but slight in late stages.

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*Klinische Untersuchungen ueber die Labyrinthæren Gleichgewichtsstœrungen mit besonderer Bereuck der allegemeiner Pruefungsmethoden und des Goniometers. *Arch. f. Ohrenh.* Bd. 78, 1909.

5. Reactions of static labyrinth partially or completely lost upon the affected side, dependent upon the extent of the area of destruction.

6. Fistula sign, that is, compression and aspiration nystagmus, absent.

IV—**Labyrinth fistula with diffuse destructive process of the labyrinth.** The symptoms and signs in this condition are the same as those of labyrinth suppuration, and briefly are:

1. Otoscopic examination shows evidence of middle ear suppuration.

2. Complete loss of hearing.

3. Absence of tinnitus.

4. Positive static labyrinth symptoms—vertigo, nystagmus and equilibrium disturbance (see former papers upon labyrinth suppuration).

5. Negative reactions from the static labyrinth of the affected side.

6. Fistula sign negative.

PROGNOSIS AND TREATMENT.

The prognosis and treatment is too large a subject to take up at the present time, besides the subject has already been considered more or less in a former paper upon the Prognosis and Treatment of Labyrinth Suppuration.

REPORT AND DISCUSSION OF A CASE OF LABYRINTH FISTULA.

WHEN comparing the history, findings and ultimate results of the present case with other similar cases of labyrinth fistula, treated after the same manner, the writer finds so much similarity that he could just as well have selected any one of the other cases.

THE CASE.*

Name—R. W.

Age—27 years.

Occupation—Clerk.

Diagnosis (made prior to and substantiated at the time of operation)—Otitis Media Suppurativa Chronica Sinistra et Cholesteatoma et Fistula Labyrinthi (external semicircular canal).

Treatment—Mastoid operation after the Zaufall method, removal of the cholesteatoma and plastic after Panse.

History—Discharge from the left ear since third year of life. Patient is unable to recall how the discharge first began or whether it was preceded by any other disease. From the third year until two years ago the discharge continued without interruption. Two months ago (June, 1907) the left ear started to discharge again. Two weeks ago the patient experienced dizziness for the first time. Every movement of the body and walking brings on an attack of dizziness which prompts the patient to seek a quiet position of the body, when the dizziness becomes less intense or disappears altogether.

With dizziness the patient experiences a sensation of turning and swaying. When questioned, he disclaims any sensation of movement of external objects. The patient has been treated by a physician prior to his admission to the hospital. He comes for treatment on account of dizziness, impairment of hearing and profuse discharge from the left ear.

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*I am indebted to Professor Alexander, of the Vienna Polyclinic Hospital, for the opportunity allowed me to study this along with the other similar cases. It was my privilege, while acting as Zimmerarzt under him, to examine, study and write up the cases before operation, to take part in the operations and to look after the after-treatment.

OTOSCOPIC FINDINGS.

Right ear normal.

Left Ear—Profuse and very offensive, dirty gray colored, purulent discharge which when wiped away shows the external canal to be normal. Complete destruction of the tympanic membrane. Remnant of the hammer handle still present. Large polyp behind the short process of the hammer. Granulations in the hypotympanum which bleed readily. Typical cholesteatoma visible. Mastoid process not sensitive and periosteum not thickened.

FUNCTIONAL EXAMINATION.

Right		Left
12 meters, +	Conv. voice	1 mete.
12 meters, +	Whisp. voice	Ad. conchr
12 meters, +	Acumeter	Ad. conch
	Weber —>	To the left
Normal	Schwabach	Shortened
+	Rinné	—
Normal	C_1,	Shortened
Normal	c_4,	Shortened
+	Watch on mastoid	—

SPONTANEOUS NYSTAGMUS.

Slight nystagmus to the right when looking to the extreme right and to the left when looking to the extreme left; but to neither side more marked than to the other.

CALORIC NYSTAGMUS.

By syringing the left ear with cold water with the head in the erect position the patient exhibits rotatory nystagmus to the right.

GALVANIC NYSTAGMUS.

Was not examined for at this sitting.

TURNING NYSTAGMUS.

After ten turnings to the left (3600°) with head inclined forward

90° the patient manifests a bilateral rotatory nystagmus to the right, when looking straight ahead, for a period of 20 seconds.

After ten turnings to the right (3600°) with head inclined forward 90° the patient manifests bilateral rotatory nystagmus to the left, lasting for a period of 20 seconds.

EQUILIBRIUM.

At the time of this examination (August 26, 1907) there was no evidence of equilibrium disturbances by the usual tests, including that made on the Alexander-Stein goniometer.

Three days later (August 29) after an accident which occurred during the local treatment, to be described later, the patient showed positive signs of equilibrium disturbances by all tests as follows:—

Rhomberg positive, gait forward and backward broad and uncertain hopping on one foot quite impossible. The Alexander-Stein goniometer test showed quite evident disturbances, as follows:—

With eyes closed and face forward the patient tends to topple over with an elevation of 14°, with face backward 11°, with face to the right 9° and with face to the left 10°.

For reasons stated below the author on this same date made the test for labyrinth fistula; as a result, compression and aspiration nystagmus or the so-called fistula symptom was found to be positive. Upon applying compression of air in the external canal of the left ear the patient manifests a pronounced horizontal nystagmus to the left and upon applying suction, a pronounced horizontal nystagmus to the right. The compression nystagmus to the left is somewhat more pronounced than the aspiration nystagmus to the right.

The examination for compression and aspiration nystagmus was not generally made prior to this time, but has been quite universally since. This case appeared at a time when aurists were beginning to recognize the importance of the symptom.

OPERATION.

The case was operated by Professor Alexander, August 29th, 1907, under general narcosis with Billroth's mixture; operation lasting 25 minutes. Operation as follows:—Typical retro-auricular incision from 4 to 5 cm. long. Laying free of the mastoid process. Chiseling open the same to the antrum which was filled with a cholesteatomatous mass. Removal of the cholesteatoma and curettement of the matrix. Thoro

removal of all granulations in the middle ear spaces. On the prominence of the external semicircular canal a 3mm. long oval shaped, fistula was found. The dura of the posterior fossa was exposed for an area about the size of a 5 cent piece. The operation was completed after the method of Zaufall; plastic after Panse; wound dressing with iodoform gauze and bandage.

August 29th. Evening of same day. Patient vomited considerably, pronounced vertigo.

August 30th. Patient vomited often, has pronounced vertigo, rotatory nystagmus to the right when looking straight ahead.

August 31. Patient feels generally better, has not vomited. Vertigo has become less severe. Rotatory nystagmus to the right when looking straight ahead, but less pronounced than yesterday.

September 1st. No vertigo. Rotatory nystagmus to the right.

September 2d. No vertigo. Rotatory nystagmus to the right.

September 3d. No vertigo. Rotatory nystagmus to the right. Slight facial palsy on left side involving all branches of the 7th nerve.

September 5th. Change of dressings. No vertigo. Rotatory nystagmus to the right present, but diminishing in intensity. Facial palsy more pronounced, patient can barely whistle and raises the left angle of his mouth poorly. Can close left eye but incompletely.

September 9th. Change of dressings. No vertigo. Rotatory nystagmus to the right less pronounced. Facial palsy unchanged. Patient feels the slightest degree of vertigo when walking. Was discharged from the hospital and made an ambulatory patient to report for further treatment and observation.

September 10th. Change of dressings. Vertigo is only present when making quick movements. Rotatory nystagmus to the right. Facial palsy unchanged. Examination of left ear with 3 meter speaking tube whispered words heard *without* failure.

September 12th. Change of dressings. Facial palsy less distinct. Patient can close left eye. Rotatory nystagmus to the right continues. Speaking tube-whispered words heard *without* failure.

September 17th. Change of dressings. Facial palsy about the same as last examination. Rotatory nystagmus to the right. When cold water is syringed into left ear the nystagmus is unaffected (not increased).

September 18th. Change of dressings. Retro-auricular wound much smaller. Facial palsy unchanged since last examination. Cold

water in left ear produces no alteration of the very slight degree of rotatory nystagmus to the right.

After ten turnings to the left with head inclined forward, pronounced rotatory nystagmus to the right lasting twenty seconds.

After ten turnings to the right with head inclined forward, rotatory nystagmus to the left lasting ten seconds.

Speaking tube, whispered voice heard *without* failure.

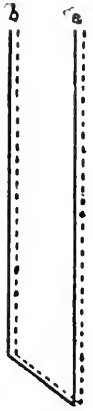
September 29th. Redressing. Facial palsy improved. Spontaneous rotatory nystagmus to the right when looking to the right. No nystagmus to the right or left when looking to the left. Same reactions to turning as on the 18th. Speaking tube, whispered voice heard *without* failure.

October 10th. Facial palsy decidedly better, other findings about the same as last examination. Wound closed and all discharge from left ear has ceased.

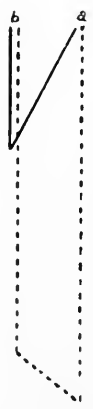
Patient was told to report at wider intervals.

A complete functional re-examination was made December 10th which gave the following results:

DECEMBER 10TH, 1907.



Right		Left
12 meters, +	Conv. voice	2½ meters
12 meters, +	Whisp voice	⅓ meter
12 meters, +	Acumeter	0
	Weber	Not lateralized
Slightly short,	Schwabach	Very short
+	Rinné	—
Normal	C^1,	0
Normal	c_4,	0
Normal	A_1	0
+	Watch on mastoid	—



SPONTANEOUS NYSTAGMUS.

When looking to the right, bilateral rotatory rhythmic nystagmus to the right. Also less marked rotatory nystagmus to the right when looking straight ahead.

No nystagmus to the left when looking to the left.

CALORIC NYSTAGMUS.

Negative, *i. e.,* syringing the left ear with large quantities of cold water does not increase the existing spontaneous nystagmus to the right.

AFTER-TURNING NYSTAGMUS.

After ten turnings to the left with head erect and with opaque glasses before the eyes, horizontal nystagmus to the right lasting 35 seconds.

After ten turnings to the right with head erect and with opaque glasses before the eyes, horizontal nystagmus to the left lasting 12 seconds.

After ten turnings to the left with head inclined forward, rotatory nystagmus to the right lasting 19 seconds.

After ten turnings to the right with head inclined forward, rotatory nystagmus to the left lasting 11 seconds.

GALVANIC NYSTAGMUS.

Kathode 4 ma. rotatory nystagmus to right increased.
Anode 10 ma. no effect.

Kathode 10 ma. no effect.
Anode 4 ma., rotatory nystagmus to right increased (?).

THREE METER SPEAKING TUBE.

Conversational voice *without failure.*

COMPRESSION AND ASPIRATION NYSTAGMUS.

Negative.

EQUILIBRIUM.

Positive evidence of equilibrium disturbances as shown by positive Rhomberg. Wide and uncertain gait both forward and backward, also side stepping and hopping on one foot less certain than normal.

ON THE GONIOMETER.

With open eyes:	*With closed eyes:*
Face forward 26°.	Face forward 14°.
Face backward 23°.	Face backward 11°.
Face to right 19°.	Face to right 10°.
Face to left 18°.	Face to left 9°.

Patellar reflexes normal as are also other deep reflexes. Vision normal, pupils react promptly to light, accommodation and convergence.

Patient's intelligence normal.

DISCUSSION OF THE CASE.

The history is that of a chronic middle ear suppuration. The fact that the discharge was intermittent suggests a cholesteatoma. The history of dizziness with the subjective sensation of turning aggravated by active movements, ameliorated by keeping quiet, in the course of chronic middle ear discharge should direct our attention immediately to the inner ear.

The dizziness, however, does not tell us the exact character of the lesion within the inner ear, for it may be due to one of several clinical conditions, an irritative lesion, circumscribed or general, or a destructive lesion, circumscribed or general.

The Otoscopic Findings. The offensive discharge in spite of the treatment, which he had prior to admission to the hospital, suggests a cholesteatoma. The complete destruction of the membrane may suggest either chronicity or marked virulence of the original infection. It more often points to the latter for in those cases of suppuration following scarlet fever we frequently find complete destruction of the membrane early. Again complete destruction of the membrane is a most favorable condition for the development of a cholesteatoma, which by the way was present too in this case (see paper by author—"Zur Clinischen Diagnostik des Mittelohrchlesteatome," Monatsshr fur ohrenheilk, 1908). A part of the cholesteatoma was visible by otoscopic examination, presenting a glistening, pearly white, smooth surface.

The granulations, bleeding readily, suggest bone involvement and they are apt to be found in conjunction with cholesteatoma.

Functional Examination. The right ear showed normal hearing function. The rooms in the hospital were not ideal for determining

hearing distances, so the figures are lower than they would otherwise have been.

The left ear showed the hearing to be greatly reduced. Lateralization of the tuning fork to the left points to middle ear affection; but since the bone conduction on the left mastoid was shortened we have a finding which points to inner ear affection. The Rinné was negative, which, when combined with shortened bone conduction, indicates middle ear disease with secondary inner ear involvement.

That C_1 was shortened points to middle ear disease.

That c_4 was shortened points to inner ear disease.

Watch on mastoid was heard on the normal side but not on the affected side, which fact points to disease of the inner ear of the affected side.

That the spontaneous, rhythmic nystagmus was present to the left when looking strongly to the left and to the right when looking strongly to the right, but to neither side more marked than to the other does *not* suggest disease of the inner ear or at least of the membranous part. On this point I wish to emphasize the fact that normal individuals when looking intently enough to the right or left side will manifest a rhythmic nystagmus in the particular direction toward which they are looking. This is purely physiologic. Again, it is possible for the osseous labyrinth to be considerably involved, especially in cases of fistula, before the membranous labyrinth shows any physical or clinical signs of involvement.

But how are we to explain the apparently contradictory facts, namely, the absence of spontaneous nystagmus and the presence of vertigo (mentioned in the history)? The vertigo mentioned in the history was evidently due to causes which were transient (circumscribed congestion) and not present at the time of the examination on the 26th of August.

That this was the case is further borne out by the results obtained thru turning.

The after-turning nystagmus was of equal duration to the two sides and approximately normal (20 seconds). Had the patient suffered an irritative lesion at the time of the examination he would have manifested a longer duration of nystagmus to the affected side. Had he suffered a destructive lesion he would have manifested a much shorter duration of the nystagmus to the affected side.

The Caloric Reaction made with cold water douched into

the left middle ear cavity indicated that the inner ear was reactive and not destroyed, for the patient showed a pronounced rotatory nystagmus to the opposite (right) side.

The Galvanic Nystagmus was not examined at this time for I had not yet worked the subject up to the point that I had later.

We come next to a very important sign in the diagnosis of labyrinthine fistula, which was present in this case, namely, compression and aspiration nystagmus called also the fistula sign.

Prior to this time it was not the routine practice to examine every case for the presence or absence of this sign, however it became so immediately afterwards.

In this case the sign was found quite accidentally. The patient after waiting three days in the hospital for his turn to be operated, began to grow anxious because of the postponement of his operation, which was made necessary on account of a rush of other cases demanding more immediate attention. In order to pacify and hold the patient in the hospital for another day it was necessary to do something. Accordingly it was agreed to remove a polyp with the snare. Upon engaging the polyp, using the average amount of pressure necessary to reach as near the base as possible, the patient suddenly cried out in terror, extended both arms, grasped the table with one hand and an assistant with the other while his face took on a wild expression with widely open eyes, and sweat flowed freely. He was a very sick looking man for a short time. I recognized it immediately as a severe attack of vertigo, the result of pressure on some part of the lateral labyrinthine wall where the capsule was defective.

After waiting several minutes until the patient had become quiet and normal again I attempted to demonstrate the presence of compression and aspiration nystagmus. Upon applying pressure the eyes made quick horizontal movements to the left (same) side, of about 6 mm. excursion, occurring at an approximate rapidity of three complete excursions for each second of time. Upon releasing the pressure and using gentle aspiration, the nystagmus changed to that of horizontal to the right of somewhat shorter excursion than it had been to the left. The character and direction of the nystagmus (remembering Ewald's experiment) established in the writer's mind the diagnosis of fistula of the osseous horizontal (external) semicircular canal with the membranous canal intact, which diagnosis was corroborated at the time of the operation.

After waiting an hour the patient was examined on the goniometer with the result that he showed diminished equilibration. Balance was lost with eyes closed at greatly reduced elevations.

The operation report shows the typical findings of a case of chronic middle ear suppuration with cholesteatoma. The cholesteatoma had by its growth caused an absorption of the bone of the prominence of the external semicircular canal; not an unusual finding in these cases. Altho there was a fistula in the osseous canal, the membranous canal had not been affected, accordingly Prof. Alexander decided to leave the labyrinth alone.

Surgically the wound did well, healing occurring completely after a reasonable length of time for such cases.

During the period of after treatment we note prominently

(a) Vertigo* which was quite pronounced immediately following the operation and for a day or so afterward. The vertigo then grew gradually less, however it was felt more or less by the patient when going around even after he had left the hospital.

This symptom taken together with the next symptom to be mentioned—spontaneous nystagmus to the right (well) side—tells us that a destructive process had occurred in the labyrinth after operation which was probably of a slower nature than a diffuse suppuration, for the patient retained hearing for at least some weeks after the operation.

(b) Spontaneous Rotatory Nystagmus to the right (well) side was present immediately following the operation and diminished gradually thereafter, but had not disappeared entirely after five months. At all times the nystagmus was demonstrable, even when the patient looked straight ahead.

The character, direction and persistency of the nystagmus found in this case is characteristic of any form of destructive lesion of the labyrinth or more exactly of that part which has to do with the static and dynamic equilibrium.

(c) Hearing was present and demonstrable with the three meter speaking tube for a considerable period after the operation, the patient having been able to detect whispered words without failure.

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*The vertigo following the operation was not due to any fault of the operation but to the uncovering of an existing fistula which had been sealed over with a cholesteatomatous mass—the vertigo was an inevitable sequence.

Eventually the hearing grew less and less until finally all hearing left the patient, he not being able to recognize conversational voice thru the tube. Furthermore he was unable to hear the new, Edelman-Bezold, small (a_1) fork that Professor Alexander had just acquired for the clinic. In short the patient grew *gradually* deaf and the deafness was *absolute*.

(d) Facial palsy developed late and as all facial palsies do which develop late after the radical operation, it disappeared early. Complete recovery occurring after a few weeks.

I made several complete examinations of the case for the purpose of study, about a month apart; however to save space I have outlined but one which was made December 10th, 1907. It is shown above in the report and really needs no further special discussion.

The patient had an inactive left labyrinth, complete deafness and demonstrable equilibrium disturbance.